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TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA

THIRD SERIES

VOLUME THE THIRTY-FOURTH

PHILADELPHIA
PRINTED FOR THE COLLEGE
1912

NOTICE

THE present volume of TRANSACTIONS contains the papers read before the College from January, 1912, to December, 1912, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WILLIAM ZENTMAYER

DORNAN, PRINTER

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1912

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[1] Died, November 19, 1912

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[1] Died April 3, 1912.

LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION

ELECTED	
1787	JOHN REDMAN
1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES[1]
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
1883	ALFRED STILLE
1884	SAMUEL LEWIS[2]
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
1889	D. HAYES AGNEW
1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.
1900	W. W. KEEN
1902	HORATIO C. WOOD
1904	ARTHUR V. MEIGS
1907	JAMES TYSON
1910	GEORGE E. DE SCHWEINITZ

[1] Died four months after his election.
[2] Resigned on account of ill-health.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA

DECEMBER, 1912

* Non-resident Fellows.
† Fellows who have commuted dues.

ELECTED

1892. Abbott, Alexander C., M.D., Sc.D. (Hon.), Professor of Hygiene and Bacteriology in the University of Pennsylvania; Member of the Board of Health of Philadelphia. 4229 Baltimore Ave.

1912. Addison, William H. F., A.B., M.B., Assistant Professor of Normal Histology and Embryology in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1905. Adler, Lewis H., Jr., M.D., Professor of Diseases of the Rectum in the Philadelphia Polyclinic and College for Graduates in Medicine; formerly Prosector to the Professor of Anatomy in the University of Pennsylvania; Consulting Surgeon to the Charity Hospital; Secretary-Treasurer of the American Proctologic Society. 1610 Arch St.

1903. Allen, Alfred Reginald, M.D. Lecturer on Neurological Electro-Therapeutics and Instructor in Neurology and Neuropathology in the University of Pennsylvania. 111 S. Twenty-first St.

1906. Allen, Francis Olcott, Jr., A.B., M.D., Surgeon to the Dispensaries of the Presbyterian, Methodist, and Pennsylvania Hospitals. 2216 Walnut St.

1896. Allyn, Herman B., M.D., Associate in Medicine in the University of Pennsylvania; Physician to the Philadelphia General Hospital. 501 S. Forty-second St.

ELECTED

1888. Anders, James M., M.D., LL.D., Professor of the Theory and Practice of Medicine and Clinical Medicine in the Medico-Chirurgical College; Consulting Physician to the Jewish Hospital Association, Philadelphia. 1605 Walnut St.

1905. Anspach, Brooke M., M.D., Associate in Gynecology in the University of Pennsylvania; Surgeon to the Gynecean Hospital; Gynecologist to the Philadelphia General and the Stetson Hospitals; Assistant Gynecologist to the University Hospital. 119 S. Twentieth St.

1905. Appleman, Leighton F., M.D., Demonstrator of Pharmacy and Materia Medica, and Instructor in Therapeutics in the Jefferson Medical College; Instructor in Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmologist to the Frederick Douglass Memorial Hospital. 308 S. Sixteenth St.

1906. Ashhurst, Astley P. C., A.B., M.D., Instructor in Surgery in the University of Pennsylvania; Assistant Surgeon to the Orthopædic Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 811 Spruce St.

1893. Ashton, Thomas G., M.D., Physician to the Philadelphia General Hospital. 1814 S. Rittenhouse Square.

1906. Babbitt, James A., A.B., (Yale), A.M. (Haverford), M.D., Professor of Hygiene and Physical Education at Haverford College; Assistant Laryngologist and Aurist, and Chief of the Out-patient Department for Diseases of the Nose, Throat, and Ear at the German Hospital; Assistant Instructor in Otology in the University of Pennsylvania; Laryngologist to the Out-patient Department of the Children's Hospital. 121 S. Eighteenth St.

1903. Bacon, John, M.D. Torresdale, Pa.

1910. Baer, Benjamin F., Jr., M.D. 2040 Chestnut St.

†1892. Baker, George Fales, B.S., M.D. 1818 Spruce St.

1911. Baldwin, James Harvey, A.B., M.D., Assistant Surgeon to the Methodist Hospital. 1426 Pine St.

1889. Baldy, John Montgomery, M.D., Professor of Gynecology in the Philadelphia Polyclinic; Surgeon to the Gynecean Hospital; Consulting Surgeon to the Jewish and the Frederick Douglass Memorial Hospitals. 2219 De Lancey Place.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home. 3709 Powelton Ave.

ELECTED

1911. Barnard, Everett P., M.D., Obstetrician to the Maternity Hospital; Assistant Instructor in Obstetrics in the University of Pennsylvania. 119 S. Nineteenth St.

1883. Baum, Charles, A.M., M.D., Ph.D. 1828 Wallace St.

1908. Beardsley, Edward J. G., M.D., L.R.C.P. (Lond.), Chief of the Out-patient Medical Department of the Jefferson Medical College Hospital; Demonstrator of Physical Diagnosis and Clinical Medicine in the Jefferson Medical College; Physician to the Byberry Branch of the Philadelphia Hospital; Assistant Physician to the Philadelphia General Hospital. 2030 Chestnut St.

1883. Beates, Henry, Jr., M.S., Sc.D., M.D. 260 S. Sixteenth St.

1874. Bennett, W. H., A.M., M.D., Physician-in-Charge of the Seashore House for Invalid Children, and of the Seaside House for Invalid Women, Atlantic City; formerly Physician to the Episcopal Hospital, and Physician-in-Charge of St. Christopher's Hospital for Children. 1837 Chestnut St.

1896. Beyea, Henry D., M.D., Associate in Gynecology and Assistant Demonstrator of Obstetrics in the University of Pennsylvania; Assistant Surgeon to the Gynecean Hospital. 1734 Spruce St.

†1884. Biddle, Alexander W., M.D. Chestnut Hill.

1884. Biddle, Thomas, M.D. 122 S. Twenty-second St.

*1903. Biggs, Montgomery H., M.D., Surgeon to the Rutherford Hospital; First Lieutenant, Medical Reserve Corps, U. S. Army; Member of the Tri-State Association of the Carolinas and Virginia; Member of the Southern Medical Association. Rutherfordton, N. C.

1908. Bland, Pascal Brooke, M.D., Chief Clinical Assistant in the Gynecological Department of the Jefferson Medical College Hospital; Instructor in Gynecology in the Jefferson Medical College; Gynecologist to St. Joseph's Hospital; Assistant Gynecologist to the Philadelphia General Hospital. 1729 Pine St.

1894. Bliss, Arthur Ames, A.M., M.D. 117 S. Twentieth St.

1894. Bochroch, Max H., M.D., Demonstrator of Neurology and Chief Clinical Assistant in the Nervous Department of the Jefferson Medical College Hospital; Neurologist to the Outpatient Department of St. Joseph's Hospital. 1539 Pine St.

1896. Boger, John A., A.M., M.D., Surgeon to St. Mary's Hospital; Surgeon to the Stetson Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 2213 N. Broad St.

ELECTED

1910. Boice, J. Morton, A.B., M.D., Gynecologist to the Out-patient Department of St. Joseph's Hospital; Lecturer on Chemistry to the Training School of St. Joseph's Hospital. 4020 Spruce St.

1911. Bonney, Charles W., A.B., M.D., Assistant Demonstrator of Anatomy in the Jefferson Medical College; Assistant Surgeon, Department of Oral Surgery, Philadelphia General Hospital. 1117 Spruce St.

1891. Boyd, George M., M.D., Professor of Obstetrics in the Medico-Chirurgical College; Obstetrician to the Medico-Chirurgical Hospital; Physician to the Philadelphia Lying-in Charity; Obstetrician and Gynecologist to the Philadelphia General Hospital. 1909 Spruce St.

1907. Boyer, Henry Percival, M.D., Physician to the Stetson Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases. 4602 Baltimore Ave.

†1884. Bradford, T. Hewson, M.D., Medical Director of the Philadelphia Life Insurance Company; Medical Director of the United Security Life Insurance and Trust Company. 1802 De Lancey Place.

1907. Bradley, William N., Ph.G., M.D., Physician to the Out-patient Department of the Children's Hospital; Assistant Physician to the Out-patient Department of the Pennsylvania Hospital; Physician to the Dispensaries of the Starr Centre. 1638 S. Broad St.

1903. Branson, Thomas F., M.D., Attending Physician to the Bryn Mawr Hospital. Rosemont, Pa.

1891. Brinton, Lewis, M.D., Physician-in-Chief to the American Hospital for Diseases of the Stomach. 1933 Spruce St.

1900. Brinton, Ward, A.M., M.D., Visiting Physician to the Tuberculosis Department of the Philadelphia General Hospital; Visiting Physician to the Hospital for Poor Consumptives at White Haven, Pa.; Visiting Physician to the Henry Phipps Institute for the Prevention, Treatment, and Cure of Tuberculosis; Visiting Physician to the Philadelphia Jewish Sanatorium for Consumptives. 1423 Spruce St.

1907. Brooks, Macy, A.B. (Princeton), M.D., Assistant Genito-urinary Surgeon to the Philadelphia General Hospital. 1314 Spruce St.

ELECTED

1887. Brubaker, Albert P., A.M., M.D., Professor of Physiology and Medical Jurisprudence in the Jefferson Medical College; Professor of Physiology and Hygiene in the Drexel Institute of Science, Art, and Industry. 105 N. Thirty-fourth St.

1851. Bullock, William R., M.D. 6439 Greene St., Germantown.

1906. Burns, Stillwell C., M.D., Lecturer on Surgery in the Medico-Chirurgical College; Assistant Surgeon to the Medico-Chirurgical Hospital. 1326 Spring Garden St.

1892. Burr, Charles W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital. 1918 Spruce St.

1906. Butler, Ralph, M.D., Professor of Diseases of the Nose and Throat in the Philadelphia Polyclinic and the College for Graduates in Medicine; Chief of the Dispensary for Diseases of the Ear at the University Hospital; Instructor in Otology in the University of Pennsylvania; Laryngologist and Aurist to the German Hospital. 1824 Chestnut St.

1912. Butt, William Redfield, M.D., Assistant Laryngologist to the Out-Patient Department of the German Hospital; Chief of the Out-Patient Department for Diseases of the Ear, Nose, and Throat of the Howard Hospital. 1701 Chestnut Street.

*1908. Cadbury, William W., A.M., M.D., University Medical School, Canton, China.

†1907. Cadwalader, Williams B., M.D., Pathologist and Clinical Assistant to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Instructor in Nervous Diseases in the Philadelphia Polyclinic. 1710 Locust St.

1905. Cameron, George A., M.D., Physician to the Germantown Hospital. S. E. cor. Schoolhouse Lane and Greene St., Germantown.

1905. Carmany, Harry S., M.D., Surgeon to St. Timothy's Hospital, Roxborough; Surgeon to the Dispensary of the Episcopal Hospital. 366 Green Lane, Roxborough.

1910. Carnett, John Berton, M.D., Associate in Surgery in the University of Pennsylvania; Assistant Surgeon to the University and the Philadelphia General Hospitals; Surgeon to the Chestnut Hill Hospital; Consulting Surgeon to the Henry Phipps Institute, to the Phœnixville Hospital, and to the Eastern Pennsylvania Institution for the Feeble-minded and Epileptic. 123 S. Twentieth St.

ELECTED

1905. Carpenter, Herbert B., M.D., Physician to the Dispensary of the Children's Hospital. 1805 Spruce St.

1895. Carpenter, John T., M.D., Lecturer on Ophthalmology in the University of Pennsylvania; Assistant Ophthalmic Surgeon to the University Hospital; Attending Ophthalmologist to the Bryn Mawr Hospital. 2040 Chestnut St.

1892. Cattell, Henry W., A.M., M.D., Editor of *International Clinics*, of *Medical Notes and Queries*, and of *Lippincott's Medical Dictionary;* Author of *Postmortem Pathology*. 3709 Spruce St.

1900. Chance, Burton, M.D., Assistant Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to the Pennsylvania Railroad Company. 235 S. Thirteenth St.

*1868. Cheston, D. Murray, M.D. Harwood P. O., Md.

1897. Cheston, Radcliffe, M.D., Visiting Physician to the Chestnut Hill Hospital; Consulting Physician to the Germantown Hospital, and to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill.

1904. Christian, Hilary M., M.D., Clinical Professor of Genito-urinary Diseases in the Medico-Chirurgical College. 1321 Spruce St.

1903. Chrystie, Walter, M.D. Bryn Mawr, Pa.

1899. Clark, John G., M.D., Professor of Gynecology in the University of Pennsylvania; Gynecologist-in-Chief to the University Hospital. 2017 Walnut St.

1897. Claxton, Charles, A.M., M.D. 5137 Morris St., Germantown.

1896. Cleveland, Arthur H., M.D., Clinical Professor of Laryngology in the Medico-Chirurgical College; Laryngologist to the Medico-Chirurgical Hospital; Laryngologist and Aurist to the Presbyterian Hospital, and to the Pennsylvania Institution for the Deaf and Dumb. 256 S. Fifteenth St.

1910. Cloud, J. Howard, M.D. 7 W. Lancaster Avenue, Ardmore, Pa.

1903. Coates, George Morrison, A.B., M.D., Professor of Diseases of the Ear in the Philadelphia Polyclinic; Chief Clinical Assistant in the Out-Patient Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Laryngologist to the Pennsylvania Institution for the Instruction of the Blind; Laryngologist to the Philadelphia Orphanage. 2032 Chestnut St.

ELECTED

1908. Codman, Charles A. E., M.D., Physician to the American Oncologic Hospital. 4116 Spruce St.

1907. Cohen, Myer Solis, A.B., M.D., Pediatrist to the Jewish Hospital, and to the Philadelphia Jewish Sanatorium for Consumptives, Eagleville, Pa.; Assistant Visiting Physician to the Philadelphia General Hospital; Consulting Physician to the Home for Consumptives, Chestnut Hill. 4102 Girard Ave.

1888. Cohen, Solomon Solis, M.D., Professor of Clinical Medicine in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital, to the Philadelphia General Hospital, to the Jewish Hospital, and to the Rush Hospital. 1525 Walnut St.

1898. Coles, Stricker, M.D., Assistant Professor of Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson and the Philadelphia General Hospitals; Visiting Physician to the Philadelphia Lying-in-Charity Hospital. 2103 Walnut St.

1901. Coley, Thomas Luther, A.B., M.D., Attending Physician to the Methodist Episcopal Hospital of Philadelphia. 338 S. Twenty-first St.

1903. Coplin, W. M. L., M.D., Professor of Pathology in the Jefferson Medical College; Pathologist to and Director of the Laboratories of the Jefferson Medical College Hospital; Pathologist to the Philadelphia General Hospital and to the Friends' Asylum for the Insane, Frankford; Bacteriologist to the Pennsylvania State Board of Health. 606 S. Forty-eighth St.

1912. Copp, Owen, A.B., M.D., Physician-in-chief and Superintendent of the Pennsylvania Hospital for the Insane. Pennsylvania Hospital for the Insane. Forty-fourth and Market Sts.

1911. Cornell, Walter Stewart, A.B., M.D., Director of Medical Inspection of Public Schools of the City of Philadelphia; Demonstrator of Osteology, and Lecturer on Child Hygiene in the University of Pennsylvania; Director of the Division of Medical Research at the New Jersey Training School for Feeble-minded Children, Vineland, N. J.; Physician to the Dispensary for Nervous Diseases of the Presbyterian Hospital. 2018 Chestnut St.

1907. Council, Malcolm S., M.D., Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Cathcart Home at Devon. Bryn Mawr, Pa.

ELECTED

*1909. Craig, Alexander R., A.M., M.D., Secretary of the American Medical Association. 535 Dearborn Ave., Chicago, Ill.

1904. Craig, Frank A., M.D., Instructor in Medicine in the University of Pennsylvania; Visiting Physician to the Henry Phipps Institute; Visiting Physician to the Free Hospital for Poor Consumptives; Physician-in-Charge of the Tuberculosis Class of the Presbyterian Hospital. 732 Pine St.

1907. Crampton, George S., M.D. 1700 Walnut St.

1904. Cruice, John M., M.D., Instructor in Medicine in the University of Pennsylvania; Assistant Physician to the Philadelphia General Hospital; Physician to the Medical Dispensary of the University Hospital and of St. Joseph's Hospital. 1815 Spruce St.

*1910. Cummins, W. Taylor, M.D., Pathologist to the Southern Pacific Hospital; Director of the Mary W. Harriman Research Laboratory, San Francisco, Cal.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1884. Curtin, Roland Gideon, A.M., M.D., Ph.D., Emeritus Physician to the Philadelphia General Hospital; Consulting Physician to the Presbyterian Hospital; Ex-President of the American Climatological Association; Ex-President of the American Society of Tropical Medicine. 22 S. Eighteenth St.

1903. Da Costa, John C., Jr., M.D., Assistant Professor of Clinical Medicine in the Jefferson Medical College; Assistant Physician to the Jefferson Medical College Hospital; Hematologist to the German Hospital; Associate Member of the Association of American Physicians. 1022 Spruce St.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General and St. Joseph's Hospitals. 2045 Walnut St.

1887. Daland, Judson, M.D., Professor of Clinical Medicine in the Medico-Chirurgical College; Physician to the Medico-Chirurgical Hospital. 317 S. Eighteenth St.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital. 5923 Greene St., Germantown.

ELECTED

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes' Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

1888. Davis, Edward P., A.M., M.D., Professor of Obstetrics in the Jefferson Medical College and in the Philadelphia Polyclinic; Visiting Obstetrician to the Jefferson and the Polyclinic Hospitals; Obstetrician and Gynecologist to the Philadelphia General Hospital; Member of the American Gynecological Society, the American Pediatric Society, and of the International Congress of Obstetrics and Gynecology. 250 S. Twenty-first St.

1889. Davis, Gwilym G., M.D. (Univ. of Penna. and Göttingen), LL.D., M.R.C.S. Eng., Professor of Orthopedic Surgery in the University of Pennsylvania; Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases; Orthopedic Surgeon to the Philadelphia General Hospital; Consulting Surgeon to St. Joseph's Hospital. 1814 Spruce St.

1900. Davisson, Alex. Heron, M.D. 31 St. Paul's Road, Ardmore.

1894. Deaver, Harry C., M.D., Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Episcopal Hospital, and to the Children's Hospital of the Mary J. Drexel Home; Surgeon-in-Chief to the Kensington Hospital for Women. 1534 N. Fifteenth St.

1887. Deaver, John B., M.D., D.Sc., LL.D., Professor of the Practice of Surgery in the University of Pennsylvania; Surgeon-in-Chief to the German Hospital. 1634 Walnut St.

1902. Dehoney, Howard, M.D. 237 S. Thirteenth St.

1885. Dercum, Francis X., A.M., M.D., Ph.D., Professor of Nervous and Mental Diseases in the Jefferson Medical College; Consulting Neurologist to the Philadelphia General Hospital; Foreign Corresponding Member of the Neurological Society of Paris, and Corresponding Member of the Psychiatric and Neurological Society of Vienna. 1719 Walnut St.

1908. Despard, Duncan L., M.D., Instructor in Surgery in Jefferson Medical College; Chief Assistant in the Surgical Clinic at Jefferson Medical College Hospital. 1806 Pine St.

1912. Dewey, J. Hiland, Ph.B., M.D., Assistant Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to St. Francis' Hospital, Trenton, N. J. 1436 Diamond St.

ELECTED

1911. Dickson, Frank D., M.D., Instructor in Orthopedic Surgery in the University of Pennsylvania; Assistant Orthopedic Surgeon to the University Hospital; Assistant Surgeon to the Orthopædic Hospital; Assistant Orthopedic Surgeon to the Philadelphia General Hospital. 1814 Spruce St.

1908. Dillard, Henry K., Jr., M.D., Physician to the Out-Patient Department of the Pennsylvania Hospital; Physician to the Dispensary of the Mary J. Drexel Home. 234 S. Twentieth St.

1891. Dixon, Samuel G., M.D., LL.D., Commissioner of Health of Pennsylvania; President of the Academy of Natural Sciences of Philadelphia; Member of the Philadelphia Bar. Bryn Mawr, Pa.

*1896. Donnellan, P. S., M.D., L.R.C.S. and P., Ireland. 4 Devonshire Place, Eastbourne, England.

*1897. Dorland, W. A. Newman, M.D., Professor of Obstetrics in the Medical Department of Loyola University; Visiting Obstetrician to Cook County Hospital; Visiting Obstetrician and Gynecologist to Jefferson Park Hospital; First Lieutenant, Medical Reserve Corps, U. S. Army. 1422 W. Jackson Boulevard, Chicago, Ill.

1907. Dorrance, George Morris, M.D., Surgeon to St. Agnes' Hospital; Demonstrator of Applied Anatomy in the Dental Department of the University of Pennsylvania. 2025 Walnut St.

1902. Dougherty, Sherborne W., M.D., Instructor in Physical Diagnosis in the University of Pennsylvania; Physician to the Dispensary of the German Hospital; Assistant Physician to the Methodist Hospital. 256 S. Sixteenth St.

1893. Downs, Norton, M.D., Fordhooke Farms, Three Tuns, Pa.

1864. Downs, R. N., M.D., Consulting Physician to the Germantown Hospital. 5916 Greene St., Germantown.

1902. Downs, Robert N., Jr., M.D., Surgeon to the Dispensary of the Germantown Hospital. 6008 Greene St., Germantown.

1910. Drayton, William, Jr., M.D., Assistant Physician to the Out-patient Department of the Pennsylvania Hospital; Clinical Assistant at the Orthopædic Hospital and Infirmary for Nervous Diseases; Physician to St. Michael's Parish School. 1316 Locust St.

1864. Duer, Edward L., A.M., M.D., Gynecologist to the Presbyterian Hospital; Consulting Obstetrician to the Maternity Hospital and the Preston Retreat. 1606 Locust St.

ELECTED

1871. Duhring, L. A., M.D., Emeritus Professor of Dermatology in the University of Pennsylvania; Honorary Member of the Dermatological Society of London, Società Itāliana di Dermatologia e Sifilographia, and Wiener Dermatologische Gesellschaft. 3322 Walnut St.

1881. Dulles, Charles Winslow, M.D., Consulting Surgeon of the Rush Hospital. 4101 Walnut St.

*1860. Dunton, William R., M.D., Montrose, Pa.

1911. Earnshaw, Henry Culp, M.S., M.D., Attending Physician to the Hospital of the Good Shepherd, Rosemont; Assistant Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Bryn Mawr Children's Hospital; Pennsylvania Railroad Surgeon. Bryn Mawr, Pa.

*1899. Edsall, David L., M.D., Jackson Professor of Clinical Medicine in the Medical School of Harvard University; Chief of Service, Medical Department of the Massachusetts General Hospital. 80 Marlborough St., Boston, Mass.

*1887. Edwards, William A., M.D., Professor of Pediatrics in the Medical Department of the University of California. Fifth and Spring Sts., Los Angeles, Cal.

1911. Eliason, Eldridge E., B.A., M.D., Assistant Instructor in Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Assistant Surgeon to the Howard Hospital; Surgeon to the Out-patient Department of the Children's Hospital. 320 S. Sixteenth St.

1904. Elmer, Walter G., B.S., M.D., Instructor in Orthopedic Surgery in the University of Pennsylvania; Assistant Orthopedic Surgeon to the University Hospital; Orthopedic Surgeon to the Jewish Hospital; Surgeon to the Pennsylvania Training School for Children. 1801 Pine St.

1896. Ely, Thomas C., A.M., M.D. 2041 Green St.

1901. Erck, Theodore A., M.D., Associate in Gynecology in the Philadelphia Polyclinic and College for Graduates in Medicine; Associate Surgeon to the Gynecean Hospital. 251 S. Thirteenth St.

1893. Eshner, Augustus A., M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic and College for Graduates in Medicine; Physician to the Philadelphia General Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Physician to Mercy Hospital. 1019 Spruce St.

ELECTED

*1905. Evans, Joseph S., Jr., A.B., M.D., Professor of Clinical Medicine and Students' Physician in the University of Wisconsin. University of Wisconsin, Madison, Wis.

1905. Evans, William, M.D. 4007 Chestnut St.

1912. Eves, Curtis C., M.D., Aural and Laryngeal Surgeon to the Episcopal Hospital; Assistant in the Out-patient Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Demonstrator of Operative Surgery of the Ear, Nose, and Throat in the Philadelphia Polyclinic. 302 S. Nineteenth St.

1894. Faries, Randolph, M.D. 2007 Walnut St.

†1903. Farr, Clifford B., A.B., M.D., Professor of Diseases of the Stomach, etc., in the Philadelphia Polyclinic; Instructor in Medicine in the University of Pennsylvania; Assistant Physician to the Philadelphia General Hospital; Pathologist to the Presbyterian Hospital. 117 S. Twenty-second St.

1893. Farr, William W., M.D., Physician to the Leamy Home. Springfield Ave. and Lincoln Drive, Chestnut Hill.

1884. Fenton, Thomas H., M.D., Ophthalmologist to St. Vincent's Home, to the Home for Aged Couples, to the Baptist Home, and to the House of the Good Shepherd. 1319 Spruce St.

1907. Ferguson, Albert D., M.D., Visiting Physician to the Widener Memorial School for Crippled Children. 50 E. Johnson St., Germantown.

1907. Fetterolf, George, A.B., M.D., Sc.D., Laryngologist to the Henry Phipps Institute for Tuberculosis; Laryngologist to the White Haven Sanatorium; Consulting Laryngologist to the Phœnixville Hospital; Demonstrator of Anatomy in the University of Pennsylvania. 330 S. Sixteenth St.

1907. Fife, Charles A., A.B., M.D., Instructor in Diseases of Children, and Associate in the William Pepper Clinical Laboratory in the University of Pennsylvania; Physician to St. Christopher's Hospital for Children, to the Children's Department of the Presbyterian Hospital, and to the Children's Department of the Mary J. Drexel Home; Assistant Pediatrist to the Philadelphia General Hospital; Visiting Physician to St. Vincent's Home. 2033 Locust St.

1884. Fisher, Henry M., M.D. 1027 Pine St.

ELECTED

1910. Fisher, John Monroe, M.D., Associate Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Philadelphia, St. Agnes', and Phœnixville Hospitals; Assistant Gynecologist to the Jefferson Medical College Hospital. 222 S. Fifteenth St.

1888. Flick, Lawrence F., M.D. 736 Pine St.

1908. Foulkrod, Collin, M.D., Obstetrician to the Maternity House of the Presbyterian Hospital; Assistant Demonstrator of Obstetrics in Jefferson Medical College; Gynecologist to the Dispensary of the Presbyterian Hospital. 4005 Chestnut St.

1908. Fox, Herbert, M.D., Director of the William Pepper Laboratory of Clinical Medicine, University of Pennsylvania; Pathologist to the Laboratory of Comparative Pathology of the Zoölogical Society of Philadelphia; Pathologist to the Rush Hospital. 3902 Locust St.

†1885. Fox, Joseph M., M.D. Torresdale, Pa.

1906. Fraley, Frederick, Jr., A.B., M.D. 1701 De Lancey Place.

1903. Francine, Albert P., A.M., M.D., Instructor in Medicine in the University of Pennsylvania; Physician to the Medical Dispensary of the University Hospital; Attending Physician to the Philadelphia General Hospital, Department of Tuberculosis; Chief Physician to the State Dispensary for Tuberculosis, Philadelphia. 1932 Spruce St.

1897. Frazier, Charles H., M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University, the Philadelphia General, and the Episcopal Hospitals; Surgeon to the Home for Crippled Children. 1724 Spruce St.

†1890. Freeman, Walter J., M.D., Emeritus Professor of Laryngology in the Philadelphia Polyclinic; Laryngologist to the Orthopædic Hospital; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb. 1832 Spruce St.

1910. Frescoln, Leonard D., M.D., Late Chief Resident Physician, and Lecturer on Chemistry to the Training School for Nurses, at the Episcopal Hospital. 3304 Baring St.

1910. Furbush, Charles Lincoln, M.D. 1501 Spruce St.

ELECTED

1889. Fussell, M. Howard, M.D., Professor of Applied Therapeutics in the University of Pennsylvania; Physician to the University Hospital, the Episcopal Hospital, St. Timothy's Hospital, and St. Mary's Hospital. 2035 Walnut St.; 421 Lyceum Ave., Roxborough.

1899. Gamble, Robert G., M.D., one of the Attending Physicians to the Bryn Mawr Hospital. Haverford, Pa.

1912. Gaskill, Henry Kennedy, M.D., Assistant Dermatologist to the Jefferson Medical College Hospital, and to the Philadelphia General Hospital. 2030 Chestnut St.

1873. Gerhard, George S., M.D., Physician-in-Chief to the Bryn Mawr Hospital; Consulting Physician to Bryn Mawr College; Consulting Physician to Villa Nova College. Fifty-eighth Street and Overbrook Ave.

1902. Ghriskey, Albert A., M.D. 3936 Walnut St.

1892. Gibb, Joseph S., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic. 1907 Chestnut St.

1899. Gibbon, John H., M.D., Professor of Surgery in the Jefferson Medical College; Surgeon to the Pennsylvania and the Bryn Mawr Hospitals; Consulting Surgeon to the Woman's Hospital. 1608 Spruce St.

1908. Gildersleeve, Nathaniel, M.D., Associate in Bacteriology in the University of Pennsylvania; Pathologist to the Bar Harbor Medical and Surgical Hospital, Bar Harbor, Me. Dormitories, University of Pennsylvania.

1897. Girvin, John H., M.D., Physician for Diseases of Women at the Presbyterian Hospital; Instructor in Obstetrics in the University of Pennsylvania. 2120 Walnut St.

1889. Githens, William H. H., M.D. 1327 Pine St.

1906. Gittings, J. Claxton, M.D., Instructor in Pediatrics in the University of Pennsylvania; Assistant Pediatric Physician to the University Hospital; Visiting Physician to the Children's Hospital of the Mary J. Drexel Home, and to the Out-patient Department of the Presbyterian Hospital. 3942 Chestnut St.

1905. Given, Ellis E. W., M.D., Surgeon to the Dispensary of the Episcopal Hospital; Surgeon to the Dispensary of the St. Christopher's Hospital for Children. 2714 Columbia Ave.

1894. Gleason, E. B., S.B., M.D., LL.D., Professor of Otology in the Medico-Chirurgical College. 2033 Chestnut St.

ELECTED

1906. Goepp, R. Max, M.D., Professor of Clinical Medicine and Dean of the Faculty of the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Visiting Physician to the Philadelphia General Hospital; Associate in Clinical Medicine in the Jefferson Medical College. 1716 Locust St.

1906. Goldberg, Harold G., M.D., Curator and Pathologist to the Wills Eye Hospital; Assistant Ophthalmic Surgeon to the Episcopal Hospital; Secretary of the Board of Trustees, House of St. Michael and All Angels; First Lieutenant, Medical Reserve Corps, U. S. Army. 1925 Chestnut St.

1893. Goodell, W. Constantine, M.D. 300 S. Thirteenth St.

1908. Goodman, Edward H., M.D., Associate in Medicine in the University of Pennsylvania; Consultant to the Medical Dispensary of the University Hospital; Assistant Physician to the University Hospital; Assistant Physician to the Philadelphia General Hospital. 248 S. Twenty-first St.

1905. Gordon, Alfred, M.D., Neurologist to the Mt. Sinai, the Northwestern General, and the Douglass Memorial Hospitals. 1430 Pine St.

†1897. Gould, George M., A.M., M.D. 401 Oriental Ave., Atlantic City, N. J.

1894. Graham, Edwin E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and the Philadelphia General Hospitals; Physician to the Franklin Reformatory Home. 1713 Spruce St.

1885. Graham, John, M.D. 326 S. Fifteenth St.

1904. Grayson, Charles P., M.D., Professor of Laryngology and Rhinology in the University of Pennsylvania; Physician-in-Charge of the Throat and Nose Department of the University Hospital; Otolaryngologist to the Philadelphia General Hospital. 1435 Spruce St.

1910. Greenman, Milton J., M.D., Sc.D., Director of the Wistar Institute of Anatomy and Biology. Wistar Institute of Anatomy and Biology, Thirty-sixth St. and Woodland Ave.

1883. Griffith, J. P. Crozer, M.D., Professor of Diseases of Children in the University of Pennsylvania; Corresponding Member of the Société de Pédiatrie de Paris; Member of the American Pediatric Society and of the Association of American Physicians. 1810 Spruce St.

ELECTED

1912. Griscom, J. Milton, B.S., M.D., Assistant Surgeon to the Wills Eye Hospital; Chief of Clinic of the Eye Dispensary of the Presbyterian Hospital. 1925 Chestnut St.

1911. Gummey, Frank Bird, M.D., Visiting Physician to the Germantown Hospital and Dispensary; Visiting Physician to the Midnight Mission. 5418 Greene St., Germantown.

1902. Gwyn, Norman B., M.D., Instructor in Medicine in the University of Pennsylvania. 23 S. Twenty-first St.

1894. Hamill, Samuel McC., M.D., Professor of Diseases of Children in the Philadelphia Polyclinic and College for Graduates in Medicine; Pediatrist to the Presbyterian Hospital; Pediatrist to St. Vincent's Home. 1822 Spruce St.

1897. Hand, Alfred, Jr., M.D., Visiting Physician to the Children's Hospital, to the Children's Hospital of the Mary J. Drexel Home, and to the Methodist Hospital. 1724 Pine St.

1886. Hansell, Howard F., M.D., Professor of Ophthalmology in the Jefferson Medical College; Ophthalmic Surgeon to the Philadelphia General Hospital and to the Jefferson Medical College Hospital; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. 1528 Walnut St.

1889. Hare, Hobart A., M.D., Professor of Therapeutics and Diagnosis in the Jefferson College; Physician to the Jefferson Medical College Hospital. 1801 Spruce St.

1903. Hart, Charles D., M.A., M.D., Physician to the Outpatient Department of the Pennsylvania Hospital; Inspector of the Eastern Penitentiary. 1317 Walnut St.

1885. Harte, Richard H., M.D., Adjunct Professor of Surgery in the University of Pennsylvania; Surgeon to the Pennsylvania and the Orthopædic Hospitals; Consulting Surgeon to St. Mary's, St. Timothy's, and the Bryn Mawr Hospitals. 1503 Spruce St.

1888. Hartzell, Milton B., M.D., LL.D., Professor of Dermatology in the University of Pennsylvania; Clinical Professor of Dermatology in the Woman's Medical College of Pennsylvania. 3644 Chestnut St.

1907. Hatfield, Charles James, A.B. (Princeton), M.D., Physician to the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis; Visiting Physician to the White Haven Sanatorium. 2008 Walnut St.

1872. Hays, I. Minis, M.D. 266 S. Twenty-first St.

ELECTED

1882. Hearn, W. Joseph, M.D., Emeritus Professor of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General Hospital. 2119 Spruce St.

1911. Heed, Charles R., M.D., Associate in Ophthalmology in the Philadelphia Polyclinic; Assistant Surgeon to the Wills Eye Hospital; Instructor in Ophthalmology in the Jefferson Medical College. 1700 Walnut St.

1908. Heineberg, Alfred, M.D., Associate in Gynecology in the Jefferson Medical College; Assistant Gynecologist to St. Agnes' and to Mt. Sinai Hospitals. 1642 Pine St.

1901. Heisler, John C., M.D., Professor of Anatomy in the Medico-Chirurgical College. 3829 Walnut St.

1884. Henry, Frederick P., A.M., M.D., Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania; Physician to the Philadelphia General Hospital; Corresponding Member of the Royal Academy of Medicine of Rome. 114 S. Eighteenth St.

1903. Henry, J. Norman, M.D., Physician to the Pennsylvania Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Assistant Physician to the Philadelphia General Hospital. 1906 Spruce St.

1891. Hewson, Addinell, A.M., M.D., Professor of Anatomy in the Philadelphia Polyclinic and College for Graduates in Medicine; Surgeon to St. Timothy's Hospital, Roxborough. 2120 Spruce St.

1909. Higbee, William S., M.D., President of the Pennsylvania State Board of Examiners for Registration of Nurses. 1703 S. Broad St.

1910. Hill, Howard Kennedy, M.D., Assistant Instructor in Medicine in the University of Pennsylvania; Physician to the Children's Medical Dispensary of the Presbyterian Hospital; Visiting Physician to the University Settlement, and to the Day Nursery; Assistant Physician to the Medical Dispensary of the Children's Hospital. 339 S. Eighteenth St.

1897. Hinkle, William M., M.D., Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory. 1323 N. Thirteenth St.

*1892. Hinsdale, Guy, A.M., M.D., Secretary of the American Climatological Association; Fellow of the Royal Society of Medicine, Great Britain; Fellow of the American Academy of Medicine; Lecturer on Climatology in the Medico-Chirurgical College, Philadelphia. Hot Springs, Virginia.

ELECTED

1888. Hirsh, A. Bern, M.D., Physician to the Home for Aged Couples. 1711 Diamond St.

1888. Hirst, Barton Cooke, M.D., Professor of Obstetrics in the University of Pennsylvania; Gynecologist to the Philadelphia General and the Howard Hospitals. 1821 Spruce St.

1903. Hirst, John Cooke, M.D., Assistant Obstetrician to the University and the Philadelphia General Hospitals; Attending Physician to the Maternity Hospital and to the Gynecological Dispensary of the Howard Hospital; Consulting Obstetrician to the Southeastern Dispensary. 1618 Pine St.

1908. Hitchens, Arthur Parker, M.D., Special Lecturer in the Medico-Chirurgical College. Glenolden, Pa.

1905. Hodge, Edward B., M.D., Surgeon to the Presbyterian and the Children's Hospitals; Surgeon to the Out-patient Department of the Pennsylvania Hospital; Surgeon to the Widener School, 346 S. Sixteenth St.

1885. Holland, James W., M.D., Sc.D., Professor of Medical Chemistry and Toxicology in the Jefferson Medical College. 2006 Chestnut St.

1906. Holloway, Thomas B., M.D., Instructor in Ophthalmology in the University of Pennsylvania; Assistant Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases; Ophthalmologist to the Pennsylvania Institution for the Instruction of the Blind; Consulting Ophthalmologist to the Henry Phipps Institute. 1819 Chestnut St.

1888. Horwitz, Orville, M.D., Professor of Genito-urinary Surgery in the Jefferson Medical College; Surgeon to the Jefferson Medical College Hospital, to St. Agnes' Hospital, and to the State Hospital for the Insane; Consulting Surgeon to the Jewish Hospital. 1721 Walnut St.

1908. Hoyt, Daniel M., M.D., Assistant Visiting Physician to the Philadelphia General Hospital. 3604 Chestnut St.

*1912. Huber, G. Carl, M.D., Professor of Histology and Embryology, and Director of the Histological Laboratory in the University of Michigan; Sometime Professor of Embryology in the Wistar Institute of Anatomy, Philadelphia; Secretary-Treasurer of the American Association of Anatomists. 1330 Hill St., Ann Arbor, Mich.

1892. Hughes, William E., M.D., Visiting Physician to the Philadelphia General Hospital; Pathologist to the Presbyterian Hospital. 3945 Chestnut St.

ELECTED

1912. Hunter, John W., B.S., M.D. 2042 Pine St.

1898. Hutchinson, James P., M.D., Surgeon to the Pennsylvania, the Methodist, the Children's, St. Timothy's, and the Bryn Mawr Hospitals; Adjunct Professor of Surgery in the University of Pennsylvania. 133 S. Twenty-second St.

1871. Ingham, James V., M.D. 1811 Walnut St.

*1885. Jackson, Edward, M.D., Professor of Ophthalmology in the University of Colorado; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic; Ophthalmologist to the Denver City and County Hospital. Metropolitan Building, Denver, Col.

1906. Jacobs, Francis Brinton, B.S., M.D., Pediatrist to the Howard Hospital; Pediatrist to the American Hospital for Diseases of the Stomach; Visiting Physician to the Philadelphia Orphan Asylum; Assistant in the Laboratory of the Polyclinic Hospital. 2032 Chestnut St.

1898. Johnson, Russell H., A.B. (Princeton), M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill, Philadelphia.

1900. Jones, Charles James, A.M., M.D., Ophthalmic Surgeon to St. Joseph's Hospital; Ophthalmic Surgeon to the House of the Good Shepherd, Germantown; Consulting Ophthalmologist to St. Vincent's Home; Assistant Surgeon to the Wills Eye Hospital. 1507 Locust St.

1900. Jopson, John H., M.D., Professor of Surgery in the Philadelphia Polyclinic; Associate in Surgery in the University of Pennsylvania; Surgeon to the Presbyterian and the Children's Hospitals. 1824 Pine St.

1900. Judson, Charles F., A.B., M.D., Physician to St. Christopher's Hospital for Children, to the Southern Home for Destitute Children and to the Sheltering Arms. 1003 Spruce St.

1902. Jump, Henry D., M.D., Instructor in Medicine in the University of Pennsylvania; Assistant Physician to the Medical Dispensary of the University Hospital. 4634 Chester Ave.

1886. Jurist, Louis, M.D. 916 N. Broad St.

1903. Kalteyer, Frederick J., M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College; Chief of the Out-patient Department, Assistant Attending Physician, and Hematologist to the Jefferson Medical College Hospital; Pathologist to the Philadelphia Lying-in-Charity. 1533 Pine St.

ELECTED

1904. Kane, Bayard, M.D. 1632 Walnut St.

*1910. Karsner, Howard T., M.D., Assistant Professor of Pathology in the Harvard Medical School. 240 Longwood Ave., Boston, Mass.

†1867. Keen, William W., M.D., LL.D., (Hon.) F.R.C.S. (Eng. and Edin.), Emeritus Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Membre Correspondant Etranger de la Société de Chirurgie de Paris; Honorary Member of the Société Belge de Chirurgie and of the Clinical Society of London; Ehren mitglied der deutschen Gesellschaft für Chirurgie. 1729 Chestnut St.

1912. Keene, Floyd E., M.D., Instructor in Gynecology in the University of Pennsylvania; Assistant Gynecologist to the University Hospital; Gynecologist to the Chestnut Hill Hospital. 116 S. Nineteenth St.

*1887. Kelly, Howard A., A.B., M.D., LL.D. (Aberdeen, Wash. and Lee, and Univ. of Pa.), Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.; Hon. Fellow of the Edinburgh Obstetrical Society, the Royal Academy of Medicine of Ireland, and of the Glasgow Obstetrical and Gynecological Society. 1406 Eutaw Place, Baltimore, Md.

1909. Kelly, James A., A.M., M.D., Surgeon to St. Mary's Hospital; Pathologist and Associate in Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine. 1621 N. Seventeenth St.

1912. Kelly, Thomas C., A.M., M.D., Assistant Instructor of Medicine in the University of Pennsylvania; Pediatrician to St. Mary's Hospital; Physician to the Out-Patient Department of the Germantown Hospital. 105 School Lane, Germantown.

1898. Kempton, Augustus F., M.D. 2118 Pine St.

1905. Kercher, Delno E., M.D. 1534 Pine St.

*1907. Kinyoun, Joseph J., M.D. (Georgetown), Professor of Pathology and Bacteriology in the George Washington University. 1423 Clifton St., Washington, D.C.

1910. Klaer, Fred Harlen, A.B., M.D., Instructor in Medicine in the University of Pennsylvania; Physician-in-Charge of the Medical Dispensary of the University Hospital; Consulting Physician to the Chester County Hospital, West Chester, Pa. 334 S. Sixteenth St.

ELECTED

1895. Kneass, Samuel S., M.D., Associate in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania. 1510 Walnut St.

1908. Knipe, Jay C., M.D., Ophthalmologist to the Jewish Hospital; Assistant Ophthalmologist to the Philadelphia General Hospital, and to the Mary J. Drexel Home; Chief of the Eye Clinic at the Jefferson Medical College Hospital; Demonstrator of Osteology and Syndesmology in the Jefferson Medical College. 2035 Chestnut St.

1908. Knowles, Frank Crozer, M.D., Assistant Dermatologist to the Philadelphia General Hospital; Dermatologist to the Northern Dispensary, the Church Home for Children, and St. Vincent's Home; Assistant Dermatologist to the Hospital of the Woman's Medical College, and to the Dispensary of the Children's Hospital; Associate Dermatologist to the Dispensary of the Howard Hospital. 332 S. Seventeenth St.

1904. Krauss, Frederick, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic and Aural Surgeon to St. Christopher's Hospital for Children and Dispensary; Ear, Nose, and Throat Physician to the Children's Seashore House for Invalid Children, Atlantic City, N. J. 1701 Chestnut St.

1905. Kremer, Walter H., M.D. 6122 Main St., Germantown.

1900. Krusen, Wilmer, M.D., Professor of Gynecology in the Medical Department of Temple University; Chief Gynecologist to the Samaritan and the Garretson Hospitals; Consulting Gynecologist to the Charity and Mercy Hospitals. 127 N. Twentieth St.

1897. Kyle, D. Braden, A.M., M.D., Professor of Laryngology in the Jefferson Medical College; Consulting Laryngologist, Rhinologist, and Otologist to St. Agnes' Hospital and to the Philadelphia Home for Incurables; Laryngologist to the New Jersey Training School for Feeble-minded Children; Bacteriologist to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1517 Walnut St.

1909. Laird, J. Packard, M.D., Visiting Physician to the Devon Branch of the Presbyterian Hospital of Philadelphia. Devon, Pa.

ELECTED

1904. Landis, Henry R. M., M.D., Visiting Physician to the White Haven Sanatorium; Member of the Staff of the Henry Phipps Institute; Assistant Visiting Physician to the Philadelphia Hospital. 11 S. Twenty-first St.

1907. Langdon, H. Maxwell, M.D., Instructor in Ophthalmology in the University of Pennsylvania; Assistant Surgeon to the Dispensary for Diseases of the Eye in the University Hospital; Assistant Ophthalmologist to the Orthopædic Hospital; Chief of the Dispensary for Diseases of the Eye of the Presbyterian Hospital. 2018 Chestnut St.

*1908. Lavenson, Ralph S., M.D. 972 Arapahoe St., Los Angeles, Cal.

1904. Le Boutillier, Theodore, M.D., Clinical Professor of Pediatrics in the Woman's Medical College of Pennsylvania; Physician to the Dispensary of St. Christopher's Hospital for Children; Pediatrist to the Woman's College Hospital Dispensary; Visiting Physician to the Baptist Orphanage and to the Southern Home for Destitute Children. 9 S. Twenty-first St.

1893. Le Conte, Robert G., A.B., M.D., Surgeon to the Pennsylvania and the Bryn Mawr Hospitals; Consulting Surgeon to the Germantown and the Gynecean Hospitals. 1530 Locust St.

1909. L'Engle, Edward M., M.D. Jacksonville, Fla.

1887. Leaman, Henry, M.D. 832 N. Broad St.

1908. Lee, Walter Estell, M.D., Surgeon to the Gynecological Dispensary of the Pennsylvania Hospital; Surgeon to the Dispensary of the Germantown Hospital; Surgeon to the Dispensary of the Children's Hospital; Surgeon to the Glen Mills School. 905 Pine St.

1903. Leffmann, Henry, A.M., M.D., D.D.S., Ph.D., Professor of Chemistry in the Woman's Medical College of Pennsylvania; Honorary Professor of Chemistry in the Wagner Free Institute of Science; Pathological Chemist to the Jefferson Medical College Hospital; Formerly Port Physician of Philadelphia. 1839 N. Seventeenth St.

1892. Leidy, Joseph, M.D., Officer l'instruction publique, France; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children. 1319 Locust St.

1877. Lewis, Morris J., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases and to the Pennsylvania Hospital. 1316 Locust St.

ELECTED

1911. Lewis, Paul A., M.D., Director of the Pathological Department of the Henry Phipps Institute of the University of Pennsylvania; Assistant Professor of Pathology in the University of Pennsylvania; Director of the Ayer Clinical Laboratory of the Pennsylvania Hospital. Henry Phipps Institute, 238 Pine St.

1904. Lindauer, Eugene, M.D., Instructor of Neurology in the Medico-Chirurgical Hospital; Associate in Clinical Medicine in the Philadelphia Polyclinic; Assistant Neurologist to the Philadelphia General Hospital. 2018 N. Thirty-second St.

1886. Lloyd, J. Hendrie, A.M. (Princeton), M.D., Neurologist to the Philadelphia General Hospital, and to the Methodist Episcopal Hospital; Consulting Neurologist to the State Asylum for the Chronic Insane at Wernersville, and to the Pennsylvania Training School for Feeble-minded Children at Elwyn. 116 S. Twenty-first St.

1907. Lodholz, Edward, M.D., Demonstrator of Physiology in the University of Pennsylvania. 3103 Diamond St.

1893. Longaker, Daniel, M.D., Surgeon to the Jewish Maternity Hospital. 1402 N. Sixteenth St.

*1906. Longcope, Warfield T., M.D., Assistant Professor of the Practice of Medicine in Columbia University, New York; Associate Attending Physician to the Presbyterian Hospital, New York. 680 Madison Ave., New York City.

1877. Longstreth, Morris, M.D. 101 Brattle St., Cambridge, Mass.

1907. Loux, Hiram R., M.D. 1614 N. Broad St.

1900. McCarthy, Daniel J., M.D., Professor of Medical Jurisprudence (George B. Wood Foundation) in the University of Pennsylvania; Neurologist to the Philadelphia General and St. Agnes' Hospitals, and to the Henry Phipps Institute. 2025 Walnut St.

1875. McClellan, George, M.D., Professor of Applied Anatomy in the Jefferson Medical College; Consulting Surgeon to the Howard Hospital. 1116 Spruce St.

1903. McConnell, Guthrie, M.D., Professor of Pathology and Bacteriology in the Medical Department of Temple University; Pathologist to the Samaritan Hospital. Temple Medical School, Eighteenth and Buttonwood Sts.

ELECTED

1895. McFarland, Joseph, M.D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College; Pathologist to the Medico-Chirurgical and to the Philadelphia General Hospitals. 442 W. Stafford St., Germantown.

1905. McKenzie, Robert Tait, A.B., M.D., Professor of Physical Education and Director of the Department of Physical Education in the University of Pennsylvania. 26 S. Twenty-first St.

*1900. McReynolds, Robert Phillips, M.D. 213 S. Broadway, Los Angeles, Cal.

1886. MacCoy, Alexander W., M.D., Fellow of the American Laryngological Association; Consulting Laryngologist to the Bryn Mawr Hospital. 216 S. Fifteenth St.

1910. MacKinney, William H., M.D., Assistant Surgeon to the Dispensary for Genito-urinary Diseases, University Hospital; Assistant in the Urological Dispensary of the German Hospital. 1701 Chestnut St.

1896. Makuen, G. Hudson, M.D., Professor of Defects of Speech in the Philadelphia Polyclinic; Laryngologist to St. Mary's Hospital and to the Frederick Douglass Memorial Hospital; Visiting Consultant on Defects of Speech to the New Jersey Training School for Feeble-minded Children. 1627 Walnut St.

1898. Marshall, George Morley, M.D., Laryngologist to the Philadelphia General Hospital; Laryngologist and Otologist to St. Joseph's Hospital. 1819 Spruce St.

1893. Marshall, John, M.D., Nat.Sc.D. (Tübingen), LL.D. Professor of Chemistry and Toxicology in the University of Pennsylvania. 1718 Pine St.

1889. Martin, Edward, M.D., John Rhea Barton Professor of Surgery in the University of Pennsylvania; Surgeon to the University, Howard, Philadelphia General, and Bryn Mawr Hospitals. 1506 Locust St.

1885. Mays, Thomas J., M.D., Visiting Physician to the Rush Hospital. 1829 Spruce St.

*1868. Mears, J. Ewing, A.M., M.D., LL.D. 1535 Land Title Building, Broad and Sansom Sts.

1911. Meigs, Edward Browning, A.B., M.D., Fellow in Physiology at the Wistar Institute. Wistar Institute, Thirty-sixth St. and Woodland Ave.

*1894. Miller, D. J. Milton, M.D., Associate Physician to the Children's Hospital. 127 S. Illinois Ave., Atlantic City, N. J.

ELECTED

1910. Miller, Morris Booth, M.D., Professor of Surgery in the Philadelphia Polyclinic; Surgeon to the Douglass Memorial Hospital; Assistant Surgeon to the Philadelphia General Hospital. 2117 Pine St.

1881. Mills, Charles K., M.D., Professor of Neurology in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital; Consulting Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1909 Chestnut St.

1904. Mitchell, Charles F., M.D., Surgeon to the Germantown Hospital and to the Out-patient Department of the Pennsylvania Hospital; Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases. 342 S. Fifteenth St.

*1888. Mitchell, John K., M.D., Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Neurologist to the Presbyterian Hospital; Attending Physician to the Pennsylvania Training School for Feeble-minded Children. 1730 Spruce St.

1856. Mitchell, Silas Weir, M.A., M.D., LL.D. (Edinburgh, Harvard, Princeton, and Toronto), Corresponding Member of the French Academy of Medicine; Corresponding Member of the Verein für innerer Medicin, Berlin; Honorary Member of the Royal Medico-Chirurgical Society of London; Doctor of Medicine, Honoris Causa, University of Bologna. 1524 Walnut St.

1908. Montgomery, Charles M., A.B., M.D., Physician to the Dispensary and Instructor in the Henry Phipps Institute (University of Pennsylvania); Physician to the Home for Consumptives at Chestnut Hill. 905 Pine St.

1882. Montgomery, Edward E., A.M., M.D., LL.D., Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Jefferson Medical College Hospital and to St. Joseph's Hospital. 1426 Spruce St.

1886. Morris, Caspar, M.D. 2050 Locust St.

1893. Morris, Elliston J., M.D., Physician to the Episcopal Hospital and to the Midnight Mission. 128 S. Eighteenth St.

1883. Morris, Henry, M.D., Professor of Anatomy in the Woman's Medical College of Pennsylvania; Senior Visiting Physician to St. Joseph's Hospital; Associate Member, Military Surgeons of U. S. A. 313 S. Sixteenth St.

1856. Morris, J. Cheston, M.D. 1514 Spruce St.

ELECTED

1906. Morrison, William H., M.D. 8021 Frankford Ave.
1897. Morton, Samuel W., M.D. 1933 Chestnut St.
1904. Moulton, Albert R., M.D., Senior Assistant Physician to the Pennsylvania Hospital for the Insane; Physician to the Out-patient Department for Nervous and Mental Diseases at the Pennsylvania Hospital; Professor of Mental Diseases in the Maine Medical School (Bowdoin College). Pennsylvania Hospital for the Insane. Forty-fourth and Market Sts.
1905. Müller, George P., M.D., Associate in Surgery in the University of Pennsylvania; Assistant Surgeon to the University and the Philadelphia General Hospitals; Surgeon to St. Agnes' Hospital; Consulting Surgeon to the Chester County Hospital. 334 S. Fifteenth St.
1905. Mutschler, Louis H., M.D., Surgeon to the Episcopal Hospital; Assistant Surgeon to the Orthopædic Hospital. 2030 Tioga St.
1896. Myers, T. D., M.D. 1521 Spruce St.

1902. Nassau, Charles F., M.D., Assistant Professor of Surgery in the Jefferson Medical College; Surgeon to St. Joseph's Hospital; Consulting Surgeon to the Frankford Hospital; Assistant Surgeon to the Jefferson Medical College Hospital. 1831 Chestnut St.
1886. Neff, Joseph S., A.M., M.D., LL.D., D.P.H. 580 City Hall.
1887. Neilson, Thomas Rundle, A.M., M.D., Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Professor of Genito-urinary Diseases in the University of Pennsylvania. 1937 Chestnut St.
1905. Newcomet, William S., M.D. 3501 Baring St.
1905. Newlin, Arthur, B.S., M.D., Physician to the Pennsylvania Hospital; Physician to the Dispensary of the Children's Hospital; Assistant Physician to the Orthopædic Haspital. 1804 Pine St.
1899. Nicholson, William Rufus, A.B., M.D., Gynecologist to the Polyclinic Hospital; Obstetrician to the Presbyterian Hospital; Associate in Obstetrics in the University of Pennsylvania. 1731 Pine St.
1889. Noble, Charles P., M.D. Gladwynne, Pa.
1898. Nolan, Edward J., M.D., Recording Secretary and Librarian of the Academy of Natural Sciences of Philadelphia. 825 N. Twentieth St.

ELECTED

1905. Norris, Charles C., M.D., Attending Physician to the Maternity Hospital; Instructor in Gynecology in the University of Pennsylvania; Assistant Gynecologist and Obstetrician to the Philadelphia General Hospital. 1503 Locust St.

1905. Norris, George William, A.B., M.D., Assistant Professor of Medicine in the University of Pennsylvania; Physician to the Pennsylvania Hospital; Assistant Physician to the University Hospital. 1530 Locust St.

*1901. Norris, Henry, M.D., Surgeon to the Rutherford Hospital. Rutherfordton, N. C.

*1865. Norris, Isaac, M.D. Fairhill, Bryn Mawr, Pa.

1892. Norris, Richard C., M.D., Lecturer on Clinical and Operative Obstetrics in the University of Pennsylvania; Obstetrician in Charge of the Preston Retreat; Visiting Obstetrician to the Philadelphia General Hospital; Gynecologist to the Methodist Episcopal Hospital, and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital. 500 N. Twentieth St.

*1884. O'Neill, J. Wilks, M.D. Bergenfield, N. J.

*1885. Osler, Sir William, Bart., M.D., Regius Professor of Medicine in Oxford University, England. No. 7, Norham Gardens, Oxford, England.

1903. Ostheimer, Maurice, M.D., Associate in Pediatrics in the University of Pennsylvania; Physician-in-Charge of the Children's Dispensary and Assistant Visiting Pediatric Physician, University Hospital; Physician to the Medical Dispensary of the Children's Hospital; Physician to the Austro-Hungarian Consulate, Philadelphia. 2202 De Lancey St.

1897. Packard, Francis R., M.D., Surgeon to the Out-patient Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Laryngologist to the Children's Hospital of Philadelphia; Consulting Aurist to the Bryn Mawr Hospital. 304 S. Nineteenth St.

1898. Page, Henry F., M.D., Assistant Physician to the German Hospital and Physician to the Medical Dispensary of the same; Instructor in Clinical Medicine in the Woman's Medical College of Pennsylvania; Physician to the Baptist Home. 1907 Girard Ave.

ELECTED

1906. Pancoast, Henry K., M.D., Professor of Röntgenology in the University of Pennsylvania and Röntgenologist to the University Hospital. Bala, Pa.

1909. Parish, Benjamin D., B.S., M.D., Assistant Instructor in Otology in the University of Pennsylvania; Assistant Surgeon to the Dispensary for Diseases of the Ear, University Hospital; Aurist and Laryngologist to St. Agnes' Hospital. 29 S. Nineteenth St.

1899. Parke, William E., M.D., Consulting Obstetrician to the Episcopal Hospital; Associate Surgeon to the Kensington Hospital for Women; Gynecologist to the Frankford Hospital. 1739 N. Seventeenth St.

1910. Patterson, Ross Vernet, M.D., Associate in Medicine and Sub-Dean in the Jefferson Medical College. 340 S. Sixteenth St.

1903. Pearce, Richard M., M.D., Professor of Research Medicine in the University of Pennsylvania. 2114 De Lancey Place.

1909. Pemberton, Ralph, M.S., M.D., Physician to the Out-patient Department of the Presbyterian Hospital. 2224 Locust St.

†1889. Penrose, Charles Bingham, M.D., Ph.D. (Harvard), Formerly Professor of Gynecology in the University of Pennsylvania. 1720 Spruce St.

†1902. Pepper, William, M.D., Dean of the School of Medicine, and Assistant Professor of Clinical Pathology in the University of Pennsylvania; Physician to the Philadelphia General Hospital; Assistant Physician to the University Hospital. 1811 Spruce St.

1912. Petty, Orlando H., A.M., M.D., Pathologist and Assistant Physician to St. Timothy's Hospital. 6215 Ridge Ave.

1905. Pfahler, George E., M.D., Director of the Röntgen Ray Laboratory of Medico-Chirurgical Hospital. 1321 Spruce St.

1907. Pfromm, George W., Ph.G., M.D. 1431 N. Fifteenth St.

1907. Phillips, Horace, M.D., Second Assistant Physician to the Pennsylvania Hospital for the Insane; Visiting Physician to the Eastern Penitentiary of Pennsylvania. Pennsylvania Hospital for the Insane, Forty-fourth and Market Sts.

1911. Physick, Emlen, M.D. Cape May, N. J.

1883. Piersol, George A., M.D., Sc.D., Professor of Anatomy in the University of Pennsylvania. 4724 Chester Ave.

1911. Piersol, George Morris, B.S., M.D., Associate in Medicine in the University of Pennsylvania; Physician to the Episcopal Hospital; Assistant Physician to the University Hospital; Registrar to the Philadelphia General Hospital. 1927 Chestnut St.

ELECTED

1905. Pitfield, Robert L., M.D., Pathologist to the Germantown Hospital; Bacteriologist to the Chestnut Hill Hospital for Lung Diseases. 5211 Wayne Ave.

1896. Posey, William Campbell, M.D., Surgeon to the Wills Eye Hospital; Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmic Surgeon to the Howard Hospital; Ophthalmologist to the Pennsylvania Hospital for Epileptics. 2049 Chestnut St.

1899. Potts, Charles S., M.D., Professor of Neurology in the Medico-Chirurgical College; Neurologist to the Philadelphia General Hospital; Consultant to the Insane Department of the Philadelphia General Hospital; Consultant to the Hospital for the Insane, Atlantic County, New Jersey. 2018 Chestnut St.

1907. Price, George E., M.D., Associate Professor of Nervous and Mental Diseases in the Jefferson Medical College; Neurologist to the Philadelphia General Hospital. 1700 Walnut St.

†1903. Pyle, Walter L., A.M., M.D., Member of the American Ophthalmological Society; Assistant Surgeon to the Wills Eye Hospital. 1931 Chestnut St.

1908. Radcliffe, McCluney, A.M. (Lafayette), M.D., Attending Surgeon to the Wills Eye Hospital. 1906 Chestnut St.

1887. Randall, B. Alexander, M.A., M.D., Professor of Otology in the University of Pennsylvania; Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb, and to St. Timothy's Hospital. 1717 Locust St.

*1904 Ravenel, Mazyck P., M.D., Professor of Bacteriology in the University of Wisconsin. Madison, Wis.

1887. Reed, Charles H., M.D. 121 S. Seventeenth St.

1885. Reichert, Edward T., M.D., Professor of Physiology in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1897. Rhein, John H. W., M.D., Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic and College for Graduates in Medicine; Neurologist to the Howard Hospital; Physician to the Philadelphia Home for Incurables; Bacteriologist to the Pennsylvania Training School for Feeble-minded Children. 1732 Pine St.

ELECTED

1906. Rhein, Robert D., M.D., Chief Physician to the Clinic of the American Hospital for Diseases of the Stomach; Assistant Physician to the Medical Dispensary of the Children's Hospital; Examining Physician to the White Haven Sanatorium. 2016 Pine St.

1891. Rhoads, Edward G., M.D. 159 W. Coulter St., Germantown.

1910. Rhoads, Samuel, M.D., Visiting Physician to the Tuberculosis Department of the Philadelphia General Hospital; Consulting Physician to the Home for Consumptives, Chestnut Hill; Physician to the Medical Dispensary of St. Christopher's Hospital for Children; Visiting Physician to the Tuberculosis Department, City Farm, Byberry, Pa. 152 Schoolhouse Lane, Germantown.

1898. Riesman, David, M.D., Professor of Clinical Medicine in the University of Pennsylvania; Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to the Philadelphia General and the Jewish Hospitals. 1715 Spruce St.

1895. Ring, G. Oram, A.M., M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmologist to the Widener Memorial Home for Crippled Children; Consulting Ophthalmologist to the American Oncologic Hospital. 2014 Chestnut St.

1905. Risley, J. Norman, M.D., Assistant Surgeon to the Wills Eye Hospital; Ophthalmologist to the Pennsylvania Training School for Feeble-minded Children. 134 S. Twentieth St.

1891. Risley, S. D., A.M., M.D., Ph.D., Attending Surgeon to the Wills Eye Hospital; Professor (Emeritus) of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Member of the Board of Managers of the Pennsylvania Training School for Feeble-minded Children; Alumnus Manager of the University Hospital. 2018 Chestnut St.

†1878. Roberts, John B., M.D., Professor of Surgery in the Philadelphia Polyclinic; Surgeon to the Methodist Episcopal Hospital. 313 S. Seventeenth St.

1899. Roberts, Walter, M.D., Professor of Otology in the Philadelphia Polyclinic; Otologist to the Methodist Episcopal Hospital; Laryngologist to the Philadelphia General Hospital. 1732 Spruce St.

1903. Robertson, William Egbert, M.D., Professor of Theory and Practice of Medicine and of Clinical Medicine in Temple University; Physician to the Episcopal, Samaritan, and Garretson Hospitals. 327 S. Seventeenth St.

ELECTED

*1910. Robinson, G. Canby, M.D., Chief Resident Physician at the Hospital of the Rockefeller Institute, Sixty-sixth Street and Avenue A, New York.

1902. Robinson, James Weir, M.D., Assistant Surgeon to the Presbyterian Hospital. 326 S. Sixteenth St.

1903. Robinson, William Duffield, Ph.G., M.D. 2012 Mount Vernon St.

1912. Rodman, John Stewart, M.D., Lecturer on Surgery in the Medico-Chirurgical College; Assistant Surgeon to the Medico-Chirurgical Hospital; Surgeon to the Dispensary of the Presbyterian Hospital; Assistant Surgeon to the Out-patient Department of the Pennsylvania Hospital. 1904 Chestnut St.

1900. Rodman, William L., M.D., Professor of Surgery in the Medico-Chirurgical College; Surgeon to the Presbyterian Hospital; Surgeon to the Philadelphia General Hospital. 1904 Chestnut St.

1909. Rosenberger, Randle C., M.D., Professor of Hygiene and Bacteriology in the Jefferson Medical College; Professor of Hygiene and Preventive Medicine in the Woman's Medical College of Pennsylvania; Director of the Clinical Laboratory of the Philadelphia General Hospital. 2330 N. Thirteenth St.

1898. Ross, George G., M.D., Assistant Surgeon to the German Hospital and Surgeon to the Out-patient Department of the same; Surgeon to the Germantown Hospital; Surgeon to the Stetson Hospital; Instructor in Surgery in the University of Pennsylvania. 1721 Spruce St.

*1907. Royer, B. Franklin, M.D. Donaldson Bldg., Harrisburg, Pa.

†1905. Rugh, James Torrance, M.D., Orthopedic Surgeon to the Methodist Hospital; Demonstrator of Orthopedic Surgery in the Jefferson Medical College; Assistant Orthopedist to the Jefferson Hospital; Assistant Orthopedist to the Philadelphia General Hospital. 1616 Spruce St.

1897. Sailer, Joseph, Ph.B., M.D., Professor of Clinical Medicine in Universtiy of Pennsylvania; Physician to the Philadelphia, the University, and Presbyterian Hospitals. 1830 Spruce St.

1900. Sajous, Charles E. de M., M.D., B.Lett., LL.D., Knight of the Legion of Honor, and Officer of the Academy of France; Corresponding Member of the Society of Public Medicine of Belgium; Member of Honor of the French Society of Hygiene, etc. 2043 Walnut St.

ELECTED

1910. Salade, Louis A., M.D., 1009 S. Oakdale Ave., Medford, Oregon.

1905. Sartain, Paul J., M.D. 212 W. Logan Square.

1908. Sautter, Albert C., M.D., Assistant in the Dispensary for Diseases of the Eye in the University Hospital; Assistant in the Eye Dispensary of the German Hospital. 1421 Locust St.

1906. Saylor, Edwin S., M.D. 2005 Chestnut St.

1910. Scarlet, Rufus B., M.D., Laryngologist to the Home for Consumptives at Chestnut Hill; Assistant in the Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Instructor in Diseases of the Nose and Throat in the Philadelphia Polyclinic and College for Graduates in Medicine. 4009 Chestnut St.

1899. Schamberg, Jay F., M.D., Professor of Dermatology and Infectious Eruptive Diseases in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Physician to the Municipal Hospital for Infectious Diseases. 1922 Spruce St.

1887. de Schweinitz, George E., A.M., M.D., Professor of Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Philadelphia General Hospital; Consulting Ophthalmic Surgeon to the Philadelphia Polyclinic; Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1705 Walnut St.

1910. Schwenk, Peter N. K., M.A., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital; Attending Surgeon to the Wills Eye Hospital. 1417 N. Broad St.

1892. Seiss, Ralph W., M.D., Professor of Otology in the Philadelphia Polyclinic; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb. 255 S. Seventeenth St.

1908. Shannon, Charles E. G., A.B., M.D., Instructor in Ophthalmology in the Jefferson Medical College; Assistant in the Ophthalmological Clinic at the Jefferson Medical College Hospital; Ophthalmologist to the Seybert Institution. 1633 Spruce St.

1897. Sharpless, William T., M.D., Physician to the Chester County Hospital. West Chester, Pa.

1906. Shields, William G., M.D., Chief of Dermatological Clinic and Assistant Physician to the Jewish Hospital. 412 Schoolhouse Lane, Germantown.

ELECTED

1890. Shoemaker, George Erety, A.M., M.D., Gynecologist to the Presbyterian Hospital and to the Pennsylvania Epileptic Hospital and Colony Farm. 1831 Chestnut St.

*1908. Shoemaker, Harlan, A.B. (Stanford), M.D. Shelby, N. C.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms; Consulting Physician to the Southern Home for Destitute Children; Assistant Physician to the German Hospital; Physician to the Out-patient Department of the German Hospital. 2011 Chestnut St.

†1896. Shoemaker, William T., M.D., Ophthalmic Surgeon to the Germantown Hospital; Assistant Ophthalmologist and Chief of Clinic to the German Hospital; Attending Surgeon to the Eye Department of the Pennsylvania Hospital; Consulting Ophthalmologist to the Pennsylvania Institution for the Deaf and Dumb, and to the Southern Home for Destitute Children. 109 S. Twentieth St.

1900. Shumway, Edward Adams, B.S., M.D., Instructor in Ophthalmology in the University of Pennsylvania, and Assistant Ophthalmic Surgeon to the University Hospital; Assistant Ophthalmologist to the Philadelphia General Hospital; Assistant Ophthalmologist and a Chief of Clinic to the German Hospital 2007 Chestnut St.

1903. Sinclair, John Falconer, M.D., Physician to the Medical Dispensary of the Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children. 4103 Walnut St.

1907. Sinkler, Francis Wharton, A.B., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Physician to the Episcopal Hospital. 1606 Walnut St.

1902. Siter, E. Hollingsworth, M.D., Instructor in Genito-urinary Diseases in the University of Pennsylvania; Genito-urinary Surgeon to the Philadelphia General Hospital; Surgeon-in-Charge of the Genito-urinary Dispensary of the University Hospital; Consulting Genito-Urinary Surgeon to the Eastern Penitentiary, and to the Philadelphia County Prison. 2038 Locust St.

1904. Skillern, Penn Gaskell, M.D. 241 S. Thirteenth St.

1904. Smith, Allen J., A.M., M.D., Sc.D. (Penna. Coll.), LL.D. (McGill Univ.), Professor of Pathology and of Comparative Pathology, and Director of Courses in Tropical Medicine in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

ELECTED

1905. Smith, S. MacCuen, M.D., Professor of Otology in the Jefferson Medical College; Aurist and Laryngologist to the Germantown Hospital; Aurist to the Jewish Hospital; Consulting Aurist to the Oncologic Hospital. 1429 Spruce St.

1908. Speese, John, M.D., Instructor in Surgery in the University of Pennsylvania; Surgeon to the Dispensary of the Presbyterian, the Children's, and the Polyclinic Hospitals. 248 S. Twenty-first St.

1895. Spellissy, Joseph M., A.M., M.D., Surgeon to the Outpatient Department of the Pennsylvania Hospital; Visiting Surgeon to St. Joseph's and to the Methodist Episcopal Hospitals; Assistant Surgeon to the Orthopedic Department of the University Hospital. 110 S. Eighteenth St.

1897. Spiller, William G., M.D., Associate Professor of Neurology and Professor of Neuropathology in the University of Pennsylvania; Clinical Professor of Nervous Diseases in the Woman's Medical College of Pennsylvania. 4409 Pine St.

1908. Spitzka, Edward Anthony, M.D., Director and Professor of General Anatomy in the Daniel Baugh Institute of Anatomy of the Jefferson Medical College. Eleventh and Clinton Sts.

1894. Stahl, B. Franklin, B.S., Ph.G., M.D., Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Lecturer on Dietetics of the Sick in the University of Pennsylvania; Visiting Physician to St. Agnes' Hospital; Visiting Physician to the Philadelphia General Hospital. 1727 Pine St.

1909. Starbuck, J. Clinton, M.D. 42E. Washington St., Media, Pa.

1875. Starr, Louis, M.D., LL.D. (Haverford). 1818 S. Rittenhouse Square.

1912. Stauffer, Nathan Pennypacker, D.D.S., M.D., Chief of the Dispensary for Diseases of the Ear, Throat, and Nose of the Presbyterian Hospital; Assistant in the Outpatient Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Aurist to the Home of the Merciful Savior for Crippled Children. 208 S. Twentieth St.

1892. Steinbach, Lewis W., M.D., Professor of Clinical and Operative Surgery in the Philadelphia Polyclinic; Visiting Surgeon to he Philadelphia General and the Jewish Hospitals. 1309 N. Broad St.

ELECTED

1910. Stellwagen, Thomas C., Jr., M.D., Chief Clinical Assistant in the Out-patient Surgical Department of the Jefferson Medical College Hospital. 110 S. Nineteenth St.

1884. Stelwagon, Henry W., M.D., Ph.D., Professor of Dermatology in the Jefferson Medical College; Honorary Fellow of the Society of Dermatology and Syphilography of Italy; Corresponding Fellow of the Society of Dermatology and Syphilography of France, of the Dermatological Society of Vienna, and of the Dermatological Society of Berlin. 1634 Spruce St.

1895. Stengel, Alfred, M.D., Sc.D., Professor of Medicine in the University of Pennsylvania; Physician to the University Hospital and the Pennsylvania Hospital. 1728 Spruce St.

1901. Stevens, Arthur A., M.D., Professor of Materia Medica, Therapeutics, and Clinical Medicine in the Woman's Medical College of Pennsylvania; Lecturer on Physical Diagnosis in the University of Pennsylvania; Physician to the Episcopal and St. Agnes' Hospitals. 314 S. Sixteenth St.

1902. Stewart, Francis T., M.D., Professor of Clinical Surgery in the Jefferson Medical College; Surgeon to the Germantown Hospital; Surgeon to the Out-patient Department of the Pennsylvania Hospital. 311 S. Twelfth St.

1898. Stout, George C., M.D., Professor of Otology in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society, and the William Penn Charter School. 1611 Walnut St.

1884. Stryker, Samuel S., M.D., Physician to the Presbyterian Hospital. 3833 Walnut St.

*1900. Swan, John M., M.D. 457 Park Avenue, Rochester, N. Y.

1898. Sweet, William M., M.D., Clinical Professor of Ophthalmology in the Jefferson Medical College, and Ophthalmic Surgeon to the Jefferson Medical College Hospital; Professor of Diseases of the Eye in the Philadelphia Polyclinic; Attending Surgeon to the Wills Eye Hospital. 1205 Spruce St.

1906. Tait, Thomas W., M.D., Ophthalmologist to the Charity Hospital. 320 S. Fifteenth St.

1900. Talley, James Ely, A.B., M.D., Physician to the Presbyterian Hospital. 218 S. Twentieth St.

ELECTED

1911. Taylor, Alonzo Englebert, M.D., Rush Professor of Physiological Chemistry in the University of Pennsylvania. 4522 Locust St.

1886. Taylor, John Madison, A.B. and A.M. (Princeton), M.D., Adjunct Professor of Non-Pharmaceutical Therapeutics in the Temple University; Editor *Monthly Cyclopedia of Practical Medicine;* Consulting Physician to the Elwyn, Pa., and the Vineland, N. J., Training Schools for Feeble-minded Children. 1504 Pine St.

1887. Taylor, William J., M.D., Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes' Hospital; Consulting Surgeon to the West Philadelphia Hospital for Women. 1825 Pine St.

1886. Taylor, William L., M.D. 1340 N. Twelfth St.

1910. Thomas, Benjamin A., A.M., M.D., Professor of Genito-urinary Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Instructor in Surgery in the University of Pennsylvania; Surgeon-in-Chief to the Out-patient Department of the University Hospital. 116 S. Nineteenth St.

1867. Thomas, Charles Hermon, M.D. 3634 Chestnut St.

1912. Thomas, Frank Wister, A.M., M.D., Visiting Physician to "Buttercup Cottage;" Consulting Physician to the Germantown Hospital. 27 E. Mt. Airy Ave.

1907. Thomas, Thomas Turner, M.D., Associate Professor of Applied Anatomy, and Associate in Surgery in the University of Pennsylvania; Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the University Hospital. 2005 Chestnut St.

1897. Thomson, A. G., M.D., Ophthalmic Surgeon to the Pennsylvania Railroad Company. 1516 Locust St.

1896. Thorington, James, A.M., M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmic Surgeon to the Presbyterian Hospital; Consulting Ophthalmologist to the American Hospital for Diseases of the Stomach; Ophthalmologist to the Ellwyn, Pa., and the Vineland, N. J., Training Schools for Feeble-minded Children. 2031 Chestnut St.

1898. Thornton, Edward Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College. 1331 Pine St.

1912. Torrey, Robert G., M.D., Assistant Physician to the Philadelphia General Hospital; Physician to the State Tuberculosis Dispensary. 255 S. Sixteenth St.

ELECTED

1896. Toulmin, Harry, M.D., Medical Director of the Penn Mutual Life Insurance Company. 925 Chestnut St.

1908. Tracy, Stephen E., M.D., Gynecologist to the Stetson Hospital. 1527 Spruce St.

1901. Tucker, Henry, M.D., Genito-urinary Surgeon to the Philadelphia General Hospital; Curator of the Academy of Natural Sciences of Philadelphia. 2000 Pine St.

†1894. Tunis, Joseph Price, M.D., Clinical Assistant to the Nose and Throat Dispensary of the Polyclinic Hospital. St. Martin's, Philadelphia.

1901. Turner, John B., M.D. 1833 Chestnut St.

1866. Tyson, James, M.D., LL.D., Emeritus Professor of Medicine in the University of Pennsylvania and late Physician to the Hospital of the University of Pennsylvania, and to the Pennsylvania Hospital. 1506 Spruce St.

1897. Tyson, T. Mellor, M.D., Physician to the Philadelphia General Hospital; Physician to the Rush Hospital, the Philadelphia Lying-in-Charity Hospital, and the Children's Aid Society of Philadelphia. 1506 Spruce St.

1904. Uhle, Alexander A., M.D., Assistant Instructor in Genito-urinary Diseases in the University of Pennsylvania; Assistant Surgeon to the Dispensary for Genito-urinary Diseases, University Hospital; Assistant Genito-urinary Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the Dispensary of the German Hospital. 1701 Chestnut St.

1907. Ullom, Josephus Tucker, M.A., M.D., Member of the Staff of the Henry Phipps Institute, Visiting Physician to the Chestnut Hill Hospital. 24 Carpenter St., Germantown.

1873. Van Harlingen, Arthur, Ph.B. (Yale), M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic; Dermatologist to the Children's Hospital. 1831 Chestnut St.

1903. Van Pelt, William Turner, M.D., Ophthalmic Surgeon to the Episcopal Hospital. 1528 Spruce St.

1893. Vansant, Eugene Larue, M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Visiting Physician to the Throat, Nose, and Ear Department of the Howard Hospital. 1929 Chestnut St.

ELECTED

1912. Vaux, Norris Wistar, M.D., Surgeon to the Chestnut Hill Hospital; Surgeon to the Dispensary of the Germantown Hospital. 8901 Germantown Ave., Chestnut Hill.

*1897. Veasey, Clarence A., A.M., M.D., Formerly Assistant Professor of Ophthalmology in the Jefferson Medical College, and Assistant Ophthalmologist to the Jefferson Medical College Hospital; Formerly Ophthalmic Surgeon to the Methodist Episcopal Hospital, Philadelphia. Suite 205, Traders' Bank Building, Spokane, Wash.

†1883. Vinton, Charles Harrod, A.M., M.D. Hotel New Chatham, Atlantic City, N. J.

1903. Wadsworth, William Scott, M.D. 207 S. Forty-fifth St.

1906. Walker, John K., M.D., Physician to the Children's Hospital of the Mary J. Drexel Home; Physician to the Out-patient Department of the Pennsylvania Hospital. 1915 Rittenhouse St.

1907. Walker, Warren, M.D., Surgeon to the Dispensary of the Episcopal Hospital; Surgeon to the Dispensary of the Children's Hospital; Assistant Surgeon in the Clinic for Genito-urinary Diseases of the Polyclinic Hospital. 1632 Spruce St.

1904. Walsh, Joseph, A.M., M.D., Visiting Physician to the White Haven Sanatorium and Vice-President of the Board of Directors; Consulting Physician to the Department of Tuberculosis at the Philadelphia General Hospital; Medical Director of St. Agnes' Hospital. 732 Pine St.

1910. Ward, E. Tillson, A.M., M.D. 1415 S. Broad St.

1893. Warren, Joseph W., M.D., Associate Professor of Physiology in Bryn Mawr College. 220 S. Broad St.

1895. Watson, Arthur W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist to the Jewish Hospital; Laryngologist and Aurist to the Mt. Sinai Hospital; Laryngologist to the Home for Incurables. 126 S. Eighteenth St.

1886. Watson, Edward W., M.D., Physician to the Magdalene Home. 38 S. Nineteenth St.

1903. Weber, Charles H., M.D., Physician to the Dispensary of the Children's Hospital. 2048 Pine St.

ELECTED

1906. Weisenburg, Theodore H., M.D., Professor of Clinical Neurology and of Neuropathology in the Medico-Chirurgical College; Neurologist to the Philadelphia General Hospital; Consulting Neurologist to the State Hospital for the Insane at Norristown, and to the State Hospital for the Feeble-minded and Epileptic at Spring City. 2030 Chestnut St.

1883. Welch, William M., M.D., Chief Diagnostician to the Bureau of Health, and Consulting Physician to the Municipal Hospital for Contagious Diseases; Consulting Physician to the Northern Dispensary and the Northern Home for Friendless Children. 1411 Jefferson St.

1897. Wells, William H., M.D., Associate in Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson Medical College Hospital. 1135 Spruce St.

1893. Westcott, Thompson S., M.D., Associate in Diseases of Children in the University of Pennsylvania; Visiting Physician to the Methodist Episcopal Hospital; Assistant Physician to the Children's Hospital; Pediatrist to the Jewish Hospital. 1720 Pine St.

1884. Wharton, Henry R., M.D., Surgeon to the Presbyterian and the Children's Hospitals; Surgeon to Girard College; Consulting Surgeon to the Bryn Mawr Hospital, the Chestnut Hill Hospital, St. Christopher's Hospital for Children, the Pennsylvania Institution for the Deaf and Dumb, and the Pennsylvania Institution for the Blind. 1725 Spruce St.

1901. White, Courtland Y., M.D., Director of the Pathological Laboratories of the Episcopal Hospital; Pathologist to the Children's Hospital and to the Kensington Hospital for Women; Chief Bacteriologist to the Bureau of Health, Department of Health and Charities, Philadelphia. 1808 Diamond St.

1878. White, J. William, M.D., Ph.D., LL.D. (Hon., Aberdeen), Fellow of the American Surgical Association and of the American Association of Genito-urinary Surgeons; John Rhea Barton Emeritus Professor of Surgery in the University of Pennsylvania. 1810 S. Rittenhouse Square.

1905. Whiteway, Harold M., M.D. 1924 Chestnut St.

1898. Whiting, Albert D., M.D., Surgeon to the Germantown Hospital; Assistant Surgeon to the German Hospital; Assistant Surgeon to the University Hospital; Instructor in Surgery in the University of Pennsylvania. 1523 Spruce St.

ELECTED

1907. Williams, Carl, B.S., M.D., Assistant Ophthalmic Surgeon to the Germantown Hospital; Assistant Surgeon to the Dispensary for Diseases of the Eye at the University Hospital. 5909 Germantown Ave.

1912. Willits, Charles H. A.M., M.D., Medical Director of the Provident Life and Trust Company of Philadelphia. Rittenhouse Hotel.

1902. Willson, Robert N., Jr., M.D., Pathologist to the Presbyterian Hospital; Assistant Physician to the Philadelphia General Hospital. 1708 Locust St.

1881. Wilson, H. Augustus, M.D., Professor of Orthopedic Surgery in the Jefferson Medical College; Emeritus Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Consulting Orthopedic Surgeon to the Philadelphia Lying-in Charity Hospital and the Kensington Hospital for Women; Senior Orthopedic Surgeon to the Philadelphia General Hospital. 1611 Spruce St.

1874. Wilson, James C., A.M. (Princeton), M.D., Emeritus Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College; Physician-in-Chief to the German Hospital; Attending Physician to the Pennsylvania Hospital; Consulting Physician to the Bryn Mawr Hospital. 1509 Walnut St.

1902. Wilson, Samuel M., M.D. 1517 Arch St.

1897. Wilson, W. Reynolds, M.D. 1709 Spruce St.

1912. Winsor, Henry, M.D., Surgeon to the Dispensary of the Episcopal Hospital; Prosector to the Associate Professor of Applied Anatomy, and Assistant Instructor in Surgery in the University of Pennsylvania. 319 S. Sixteenth St.

1904. Wister, James W., M.D., Physician to the Out-patient Department of the Germantown Hospital. 5430 Germantown Ave.

*1901. Witmer, A. Ferree, M.D. Freeport, Long Island, N. Y.

1893. Wood, Alfred C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Surgeon to the University, the Philadelphia General, St. Timothy's, and the Howard Hospitals. 2035 Walnut St.

1900. Wood, George B., M.D., Instructor in Laryngology in the University of Pennsylvania; Assistant in the Clinic for Diseases of the Nose and Throat at the Polyclinic Hospital. 129 S. Eighteenth St.

ELECTED

1865. Wood, Horatio C., M.D., LL.D. (Yale and Lafayette), Emeritus Professor of Materia Medica and Therapeutics in the University of Pennsylvania; Associate Fellow in Medicine and Surgery of the American Academy of Arts and Sciences; Member of the National Academy of Science. 4107 Chester Ave.

1903. Wood, Horatio C., Jr., M.D., Professor of Pharmacology and Therapeutics in the Medico-Chirurgical College. 1905 Chestnut St.

1880. Woodbury, Frank, M.D., Secretary to the Committee on Lunacy of the Board of Charities of Pennsylvania. 218 S. Sixteenth St.

*1911. Woods, Andrew H., A.B., M.D., Neurologist to the Canton Hospital and to the Canton Hospital for Women; Acting President and Medical Superintendent of the Canton Christian College. Canton Christian College, Canton, China.

1901. Woods, Richard F., M.D., Assistant Surgeon to the Gynecean Hospital. 1501 Spruce St.

†1897. Woodward, George, M.D. W. Willow Grove Ave., Chestnut Hill, Philadelphia.

1903. Worden, Charles B., M.D., Associate in Diseases of the Stomach and Intestines in the Philadelphia Polyclinic; Physician to the Dispensary of the Presbyterian Hospital; Anesthetizer to the Orthopedic Department of the University Hospital; Physician to the Presbyterian Orphanage. 322 S. Sixteenth St.

1889. Young, James K., M.D., Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Associate Professor of Orthopedic Surgery in the University of Pennsylvania. 222 S. Sixteenth St.

1894. Zentmayer, William, M.D., Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Attending Surgeon to the Wills Eye Hospital; Ophthalmologist to the Glen Mills School. 1819 Spruce St.

1899. Ziegler, S. Lewis, A.M., M.D., Sc.D., Attending Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to St. Joseph's Hospital; Membre Société Française d'Ophtalmologie. 1625 Walnut St.

1887. Ziegler, Walter M. L., A.M., M.D. 1418 N. Seventeenth St.

1895. Zimmerman, Mason W., M.D., Consulting Ophthalmic Surgeon to the Germantown Hospital. 1522 Locust St.

ASSOCIATE FELLOWS

(Limited to Fifty, of whom Twenty may be Foreigners)

American

ELECTED

1911. Abbe, Robert, M.D. 13 W. Fiftieth St., New York City, N. Y.

1909. Billings, Frank, M.D., 335 E. Twenty-second Street, Chicago, Illinois.

1876. Billings, John S., M.D., U. S. A., Astor Library Building, 40 Lafayette Place, New York City, New York.

1877. Chaille, Stanford E., M.D., University Building, New Orleans, Louisiana.

1886. Cheever, David W., M.D., 557 Boylston Street, Boston, Massachusetts.

1893. Councilman, William T., M.D., Harvard Medical College, Boston, Massachusetts.

1909. Crile, George W., M.D., 1021 Prospect Avenue, S. E., Cleveland, Ohio.

1909. Dana, Charles Loomis, M.D., 53 West Fifty-third Street, New York City, New York.

1892. Emmet, Thomas Addis, M.D., 91 Madison Avenue, New York City, New York.

1892. Fitz, Reginald H., M.D., 81 Arlington Street, Boston, Massachusetts.

1903. Gorgas, William C., M.D., U. S. A., Washington, D. C.

1891. Jacobi, A., M.D., 19 East Forty-seventh Street, New York City, New York.

1895. McBurney, Charles, M.D., 28 West Thirty-seventh Street, New York City, New York.

1912. McCaw, Lieut. Col. Walter D., M.D. Care of Surgeon-General's Office, Washington, D. C.

1909. Mall, Franklin P., M.D., 1514 Bolton Street, Baltimore, Maryland.

ELECTED

1906. Mayo, William J., M.D., Rochester, Minnesota.
1906. Pilcher, Lewis Stephen, M.D., 386 Grand Avenue, Brooklyn, New York.
1886. Reeve, John C., M.D., LL.D., S. W. corner Third and Wilkinson Streets, Dayton, Ohio.
1906. Shattuck, Frederick C., M.D., 135 Marlborough Street, Boston, Massachusetts.
1896. Sternberg, George M., M.D., U. S. A., 1019 Sixteenth Street, N. W., Washington, D. C.
1896. Tiffany, L. McLane, M.D., 831 Park Avenue, Baltimore, Maryland.
1909. Wadsworth, Oliver F., M.D., 526 Beacon Street, Boston, Massachusetts.
1894. Warren, J. Collins, M.D., 58 Beacon Street, Boston, Massachusetts.
1894. Weir, Robert F., M.D., 11 East Fifty-fourth Street, New York City, New York.
1892. Welch, William H., M.D., Johns Hopkins Hospital, Baltimore, Maryland.

Foreign

1890. Baccelli, Guido, Rome, Italy.
1908. Bannerman, W. B., M.D., Lt. Col. I. M. S., 11 Strathearn Place, Edinburgh, Scotland.
1894. Brunton, Sir T. Lauder, M.D., 10 Stratford Place, London, W., England.
1903. Finlay, Charles J., M.D., Havana, Cuba.
1899. Fraser, Sir Thomas R., M.D., LL.D., F.R.C.P., F.R.S., 13 Drumsheigh-Gardens, Edinburgh, Scotland.
1903. Horsley, Sir Victor Alexander Haden, F.R.S., 25 Cavendish Square, London, England.
1906. Hutchinson, Jonathan, M.D., LL.D., F.R.S., 15 Cavendish Square, London, W., England.
1896. Jaccoud, Prof. S., Rue Tronchet 35, Paris, France.
1894. Jackson, J. Hughlings, M.D., 3 Manchester Square, London, England.
1893. von Jaksch, Rudolf, M.D., Prague, Bohemia.
1903. Kocher, Prof. Theodor, M.D., Berne, Switzerland.
1912. Lazarus, Paul, M.D., Hindersinstr. 14, Berlin, N. W., Germany.

ELECTED

1909. Macallum, Archibald B., M.A., M.B., Ph.D., Sc.D., LL.D., F.R.S., 59 St. George Street, Toronto, Canada.

1906. Myles, Sir Thomas, M.D., 33 Merion Square, W., Dublin, Ireland.

1896. Pye-Smith, P. H., M.D., 48 Brook St., London, W., England.

1898. Roddick, Thomas G., M.D., 80 Union Avenue, Montreal, Canada.

1908. Ross, Major Ronald, M.D., R.A., Liverpool School of Tropical Medicine, Liverpool, England.

1869. Valcourt, Th. de, M.D., Cannes, France.

1904. Waldeyer, Prof. Wilhelm, M.D., Berlin, Germany.

CORRESPONDING MEMBERS

ELECTED

1880. Carrow, Flemming, M.D., Washington Arcade, Detroit, Michigan.

1880. Chiara, Domenico, M.D., Florence, Italy.

1886. Dey, Kanny Lall, M.D., Calcutta, India.

1885. Rendu, Jean, M.D., Lyons, France.

NECROLOGICAL LIST

FELLOWS

ARTHUR V. MEIGS,	January 1, 1912
GEORGE FRIEBIS,	January 26, 1912
JOHN H. MUSSER,	April 3, 1912
JOHN W. LODGE,	May 4, 1912
T. HEWSON BACHE,	July 8, 1912
RICHARD A. CLEEMANN,	November 19, 1912
WILLIAM R. HOCH,	December 7, 1912

ASSOCIATE FELLOWS

LORD LISTER,	February 11, 1912
ROBERT FLETCHER,	November 8, 1912

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REMARKS BY FELLOWS OF THE COLLEGE ON THE ANNOUNCEMENT BY THE PRESIDENT OF THE DEATH OF DR. ARTHUR V. MEIGS JANUARY 3, 1912

Dr. S. Weir Mitchell.

The president has announced to you, as a matter of form, the lamentable death of our Fellow and late president, Arthur V. Meigs. It has naturally fallen to me, as the oldest ex-president of the College, to speak of our late fellow member.

One of the few privileges of age is to have come into possession of what I might call inherited friendships. I have been in intimate relation with three generations of Dr. Meigs' family—grandfather, father, and son. It is therefore with more than usual reason that I took permission to put on paper in the quiet of my home what I could scarcely trust my tongue to say to you here, for to me as to many of you this loss must be one of rather unusual quality.

None of you can fail to remember the perfect courtesy with which Dr. Meigs presided over this body during the long period of grave differences of opinion when we were so fortunate as to have him at our head.

His relation to the College, to science, and to the practice of our profession will be left to other hands than mine for commemoration. All that I can do now is to express what I know are your feelings and my own regrets for the death of this much-loved physician who for many years has been my personal friend. He had a group of qualities which insured respect and justified affection, his high ideals of honor and a certain sweetness of character being felt throughout his life. In fact, no difference of opinion as to matters scientific or connected with the affairs of the College

ever in the slightest degree disturbed his constantly amiable relation to men.

With these few words, which inadequately express my own feelings, I simply leave him to the more complete characterization which he will receive when a record of his life is laid before the College.

Dr. J. C. Wilson.

After what Dr. Mitchell has said it seems to me that there is very little for me to say at the present time. Still, at an hour like this, when almost suddenly a colleague with whom we of the Pennsylvania Hospital sat in the discharge of our duties as members of the Staff a few days ago, has been called away, and particularly when one who has made such an impression upon the life of his day has thus passed from among us, one feels impelled to express the thoughts which arise in his mind. Dr. Meigs not only had an engaging personality, but he had remarkable qualities in other respects. It seems to me that if one were to characterize him it would be to say that probity, clearness of thought, tenacity of purpose were characteristics. Of his ability there could be no question. Early in his medical career, very early, he was chosen a member of the staff of the Pennsylvania Hospital to succeed his father. Comparatively early, and at an age when men are not often selected for that position, he was chosen a member of the Board of Trustees of the University of Pennsylvania. In these positions and many others he exercised those peculiar gifts of character to which I have alluded as being specially developed in him to a high degree—straightforwardness, absolute scorn of anything that was not exactly as represented, clearness of thought —not always seeing things as others saw them, but seeing them very clearly as he saw them, and he brought these qualities to bear on all his work.

Dr. Meigs belonged to a distinguished succession of medical men—distinguished not only in this community, but far beyond its limits. It was particularly, however, in his relations to this College that Dr. Meigs showed, I think, his ability and the finer

qualities of his character. Having once served the College in many capacities, it was during a time when real honest differences of opinion existed among the Fellows of the College and questions of vital interest came up for discussion that he gave to the College special service. Dr. Meigs was at that time President, and by his admirable administration he influenced the development of thought as between what might be called the conservatives and the radicals in such a manner as to bring the questions pending to successful issue. Temperamentally he was a conservative, but the sort of a conservative who proves useful in all broad and important movements—the honest, clear-headed conservative, holding in check the more decided and emotional radical until matters in question can be thoroughly threshed out, so that his very conservatism proved a source of great usefulness to this institution. Then, of course, one does not speak of the personal friendships lightly. Nothing can be added to what Dr. Mitchell has said of Dr. Meigs. We all loved him. More than that, we all held him in the highest esteem and respect.

MEMOIR OF ALOYSIUS OLIVER JOSEPH KELLY, M.D.[1]

By JAMES TYSON, M.D.

Aloysius Oliver Joseph Kelly was born June 13, 1870, and died February 23, 1911, aged forty-one years. He was chosen a Fellow of the College in 1897.

Of his boyhood I have been able to learn but little, but as the boy is father to the man it is not difficult to picture the youth of him whom we knew from early manhood to middle life. He would be somewhat delicate in appearance, quiet in speech, and gentle in manner, but a close observer withal. So, too, we may infer mental precocity in one who took the A. B. degree when eighteen, was a master of arts at his college, La Salle, at twenty-one, and a doctor of medicine from the University of Pennsylvania the same year, 1891. As became the ambitious young graduate in medicine, he entered a hospital (the St. Agnes) as resident physician in July the same year. The years 1892–94 found him a student abroad, chiefly at Vienna, where he remained two years, but also at London, Dublin, Prague, and Heidelberg. Among those with whom he worked were Chvostek, Weichselbaum, Kretz, and Paltauf.

Soon after his return he was appointed recorder in the medical dispensary of the University Hospital, where he served 1894–97. His promotion was rapid, and in 1896 he became an instructor in physical diagnosis in the University. In 1897 he again went abroad for a time. In 1899 he was made an instructor in clinical

[1] Read January 3, 1912.

medicine, and in the same year assistant physician to the University Hospital. In 1903 he was appointed an associate in medicine, and in 1906 assistant professor of medicine in the University, a position he held at the time of his death. He was appointed physician to St. Mary's Hospital in 1895, and director of the laboratory of the Philadelphia Polyclinic the same year; pathologist to St. Agnes' Hospital in 1894, and physician to the same in 1897; professor of the theory and practice of medicine in the University of Vermont in 1900, professor of pathology in the Woman's Medical College in 1906, and at the same time chief pathologist to the hospital of that college.

Dr. Kelly early became a contributor to medical journals. Among his first papers was one entitled "Advantages Presented by London to American Medical Students," in the *International Medical Magazine* for May, 1895, followed by another on "Opportunities Offered by Prague, Heidelberg, and Dublin to American Medical Students," in the *International Medical Magazine*, vol. ix. Others followed in rapid succession, and a list of more than thirty-three papers on major subjects is before me, of which I name a few by title:

"The Diagnosis and Treatment of Incipient Locomotor Ataxia," *International Clinics*, vol. ii, seventh series.

"Ueber Hypernephrome der Niere," *Beiträge zur path. Anat. und allgem. Path.*, vol. xxiii, and later in the *Philadelphia Medical Journal*, July, 1898.

"Clinical Significance of Pulsation in the Veins," *Philadelphia Polyclinic*, September 3, 1898, vol. ii.

"The Histology and Histogenesis of Certain Tumors of the Parotid Gland, with Special Reference to Those of Endothelial Origin," *Philadelphia Monthly Medical Journal*, February, 1899.

"Acute Lymphocytic Leukemia, with Reference to its Myelogenous Origin," *University of Pennsylvania Medical Bulletin*, October, 1903.

"Nature and Lesions of Cirrhosis of the Liver," *American Journal of the Medical Sciences*, December, 1905.

"Multiple Serositis—The Association of Chronic Obliterative Pericarditis with Ascites," *Transactions of the College of Physicians,* Philadelphia, 1902.

"Infections of the Biliary Tract," Mütter Lecture at the College of Physicians of Philadelphia, 1905; published also in the *American Journal of the Medical Sciences,* September and November, 1906.

He was editor of the *International Clinics* from 1903 to 1907, and of the *American Journal of the Medical Sciences* from 1907 to the date of his death. In both these positions he was accurate and painstaking, profiting greatly by his experience and developing in ability and influence. Notably as pathologist to the German Hospital he recorded a large number of autopsies and accumulated a corresponding amount of pathological knowledge. It is literally true that thousands of specimens of diseased appendices passed under his observation, while opportunities for the study of morbid anatomy in many directions were intelligently availed of. From a study of lesions of the liver resulted his Mütter Lecture, delivered at the College of Physicians in 1905, on "Infections of the Biliary Tract." The same may be said of his contribution to Osler's *System of Medicine,* an exhaustive paper covering diseases of the liver, gall-bladder, and biliary ducts.

As a writer Dr. Kelly was clear and emphatic. He had the courage of his opinions and spoke them freely. His selection as editor first of the *International Clinics* and later of the *American Journal of the Medical Sciences* was the result of the deliberate judgment of those trained to select able journalists. While performing the duties of these positions he further improved his own style and developed acuteness in the recognition of able and well-prepared papers. Contributions to his journals not up to the standard he unhesitatingly declined. Such literary discipline and accumulated knowledge of medicine prepared him for his most ambitious effort, a text-book on the *Practice of Medicine,* published by Lea & Febiger in 1910. It was finished a few months before his death, and would without doubt have added to his reputation and income had his life

been spared. Unfortunately a book on any medical subject of a practical nature dies with its author.

Without ostentation he was a judge of good literature, and though his general library was not large it was well selected. It included such books as *Maximes de Rochefoucald* and *Practical Wisdom*, embodied in the classic essays of Francis Osborn, Sir Walter Raleigh, Lord Burleigh, Sir Matthew Hale, William, Earl of Bedford, and Sir George Savile, variously written between 1617 and 1688. Copies of these books found in his library are marked and scored only as a thoughtful and appreciative reader would mark them.

The following quotations from Rochefoucald are a few of many thus marked, indicating the cast of his thoughts and reflections:

"Little minds are too much hurt by little things; great minds perceive them and are not touched by them."

"Humility is the true proof of Christian virtue; without it we retain all our faults, and they are only hidden by pride, which conceals them from others and often from ourselves."

In Sir George Savile's "Advice to a Daughter" he marked:

"These affected appearances are ever suspected, like very strong perfumes, which are generally thought no very good symptoms in those that make use of them."

"It is not true devotion to put on an angry zeal against those who may be of a differing persuasion."

"Religion is exalted reason, refined and sifted from the grosser parts of it. It dwelleth in the upper region of the mind, where there are fewest clouds or mists to darken or offend it."

Less is known than should be of Dr. Kelly's work at Burlington, Vermont, as professor of the theory and practice of medicine in the University there. I have asked his intimate friend and colleague, Dr. A. F. A. King, of Washington, D. C., professor of obstetrics, to write some impressions of Dr. Kelly growing out of his close associations with him in that faculty. He says:

"Dr. Kelly became professor of the theory and practice of medicine in the University of Vermont in 1900. During his ten years of service in this institution, from the date of his election

to his lamented decease, in 1911, he greatly endeared himself to his colleagues in the Faculty and to the students under his tuition.

"In his own special subject of practical medicine he greatly improved the methods of instruction, particularly in the matter of clinical teaching. He was the first to introduce the method of teaching by 'conference,' wherein a limited number of students were required to examine a case, and confer with each other as to the diagnosis, prognosis, and treatment, any errors being explained and corrected before the class by Dr. Kelly himself. Thus the students were really taught to 'practise medicine' before their graduation. This method, although now almost universal, was first introduced into the University of Vermont by Dr. Kelly.

"His regular lectures (other than clinical), usually read from manuscript, were carefully prepared, dignified, and finished productions, liberally interpolated with illustrative cases verbally related, which on the whole reminded some of us of the famous discourses in Sir Thomas Watson's *Practice of Physic*, read and enjoyed by so many students fifty years ago.

"Engrossed with his subject, forgetful of himself and apparently to applause, his manner in the lecture room was a happy combination of quiet dignity, earnestness, and enthusiasm. Furthermore, his lectures were remarkably exempt from that ostentatious description of incidents in his own practice, which usually contribute to the magnification of the speaker rather than to the student's edification. Dr. Kelly preferred to prove the value of his methods of treatment by results obtained in thousands of cases throughout the hospitals of the world rather than by voluble descriptions of a few isolated cases of his own.

"In fact, it may be truthfully said that in all the duties of his professional life Dr. Kelly was actuated by ethical principles of the highest type. True to himself and his convictions, as well as considerate of others, he was always fearless in the expression of his views.

"Whether in the lecture room, the examination room, or the hospital, his relations with college officials, professors, students,

nurses, and patients were characterized by honor, fidelity, and fearlessness.

"Personally, everyone who knew Dr. Kelly at all intimately could not fail to be happily impressed with the kindliness and sympathy of his gentle and tender disposition, and no one could fail to love him in return."

Dr. Kelly was chosen a Fellow of the College in 1897. He served the College in the capacity of counsellor, a member of the hall committee, member of the committee on the Alvarenga prize, and chairman of the committee on scientific business. He performed the duties of these positions with his usual fidelity, but it was as chairman of the committee on scientific business that he rendered most service. His journalistic training, and especially his experience in judging of the merits of medical papers and the personal qualification of authors, fitted him peculiarly for this service. Soon after his incumbency our *Transactions* began to show the effects of his ability in the selections of subjects and their authors, while the papers grew in number and variety and improved in quality.

Dr. Kelly's conduct while under the spell of his mortal illness was heroic, and the picture of his life as illuminated by our later knowledge was pathetic. Many of his friends realized that he was ailing, but only one or two were aware that he was the conscious victim of a disease which at his age was sure to be fatal in a few years. With his secret locked in his breast, he continued not only to exact of himself a conscientious discharge of his own duties, abating no jot or tittle in their fulfilment, but was always ready to help others who sought his assistance.

Dr. Kelly was a consistent member of the Roman Catholic Church. Among his intimates were such men as Archbishop Ryan and Monsignor Turner, the late Right Rev. John W. Shanahan, Bishop of Harrisburg, and the Rev. Francis P. Fitzmaurice, of Frankford, Philadelphia.

Among his papers was found the following typewritten definition of a gentleman from a source unknown to me. Perhaps some Fellow of the College may be able to locate it:

"A *Gentleman:* A man who is clean inside and out; who looks neither up to the rich nor down to the poor; who can lose without squealing and win without bragging; who is considerate of women, children, and old people; who is too brave to lie, too generous to cheat; who takes his share of the world and lets others have theirs."

All of these attributes Dr. Kelly possessed, though they were not needed, his appearance and deportment alone furnishing the guinea stamp.

"Though modest, on his unembarrassed brow
Nature had written—gentleman."

MEMOIR OF JOHN BEDFORD SHOBER, M.D.[1]

By CHARLES B. PENROSE, M.D.

John Bedford Shober was born in Philadelphia August 28, 1859. He was the eldest son of Samuel Lieberkuhn Shober and Ann Bond Cochran Shober. His early education was received at the formerly well-known Fairies' School in Philadelphia and at St. Paul's School in Concord, New Hampshire. He entered Princeton College in 1878 and graduated with the degree of A. B. in 1882. At Princeton he was a good student and an all-round athlete, taking an active part in rowing, football, and sparring.

John Shober came of distinguished medical ancestors, being descended on his father's side from Lieberkuhn, physician at the court of Frederick the Great, and from Dr. Samuel Shober, of Philadelphia, who died of yellow fever during the epidemic of 1793. On his mother's side he was descended from Dr. Phineas Bond—who with his brother, Dr. Thomas Bond and Dr. Zachary, was instrumental in founding the Pennsylvania Hospital. From boyhood Dr. Shober had shown a taste for medicine, and upon leaving Princeton he entered the Medical School of the University of Pennsylvania and graduated in 1885. He was resident physician at the Children's Hospital for some months after graduation, and at the Pennsylvania Hospital from February 1, 1886, to June 1, 1887. Upon leaving the Pennsylvania Hospital he spent two years abroad, studying pathology, bacteriology, and surgery, under Virchow, Koch, Bergman, and Czerney.

Returning to Philadelphia in 1889, he began the practice of his profession, in which he was active until his death. Dr. Shober was

[1] Read March 6, 1912.

a member of most of the local medical societies of Philadelphia and of the American Röntgen Ray Society. He was elected to the College of Physicians in 1891.

He was surgeon of the First Troop of Philadelphia City Cavalry from 1892 to 1895, and for several years was one of the local examining surgeons for pensions under the Federal Government.

Dr. Shober's tastes led him into surgery and surgical gynecology, and he was one of the group of men in Philadelphia who first really practised antiseptic and aseptic surgery, and who, following the teachings of Tait, successfully opened the abdomen for pelvic inflammatory disease in women. Shober's first celiotomy for pyosalpinx was in 1886. He was the first surgeon in Philadelphia deliberately to remove the appendix in the interval between attacks. This operation was performed November 11, 1889, and was, I believe, the third of the kind in this country, two similar operations having been previously performed by Nicholas Senn.

Dr. Shober continued his work in abdominal surgery and gynecology until his death. He was connected with the Gynecean Hospital almost from its foundation; he was gynecologist and surgeon at the Howard Hospital and the American Hospital for Diseases of the Stomach, and was obstetrician to the Philadelphia General Hospital from 1897 to 1905.

In the last years of his life he had been much interested in the application of Röntgen rays and radium in medicine, and had spent much time and thought in his investigations of this subject. He owned enough radium to work with, and had a thorough knowledge of all that had been done by his fellow workers in this branch of medicine. He was an enthusiastic believer in the value of radium as a therapeutic agent, and had made several ingenious devices for obtaining radium emanations for therapeutic purposes. Immediately before his death he had planned a number of experiments that, unfortunately, were never to be performed. On the subject of radium therapy he was one of the few experts in this country.

In 1895 Dr. Shober married Miss Margaret S. Harlan, daughter of Dr. George C. Harlan, a distinguished ophthalmologist and a

Fellow of this College. Two children, Margaret and Anthony Morris, and their mother survive him.

Dr. Shober died April 27, 1912, after operation for an obscure abdominal trouble.

By his death we have lost a genial and loyal comrade, whose motives were always honorable, and who aspired to the best ideals of his profession.

MEMOIR OF JOHN HERR MUSSER, M.D.[1]

By GEORGE A. PIERSOL, M.D.

The colony established by William Penn on the Delaware, governed as it was by principles insuring religious liberty to all, offered a tempting shelter from oppression and persecution. Among those early taking advantage of this security were the Swiss Mennonites, whose simple but high standards of belief and living incurred the disapproval of a dominant orthodox church. Securing by purchase from Penn, in 1710, a tract of some 10,000 acres, these Swiss pioneers began the conquest of the wilderness where now stands the city of Lancaster, Pennsylvania.

Among these settlers was Dr. Benjamin Musser, a native of Bern, who not only acquired land, but also received permission from the Proprietor to practise his accredited profession of physic. From this ancestor arose a family which for generations has been closely identified with medicine.

John Herr Musser, the subject of this memoir, was born on June 22, 1856, at Strasburg, Lancaster County, Pennsylvania. He was the son of Dr. Benjamin and Naomi Musser. In addition to his father, Dr. Musser's paternal grandfather and great-grandfather were practitioners of medicine. His maternal grandfather was Bishop John Herr, of the Mennonite church, also of Swiss descent.

Succeeding his preparatory education at the High School of Strasburg and the usual classical course at the Millersville State Normal School, Dr. Musser fulfilled the traditions of his family by studying medicine. He entered the medical department of

[1] Read November 6, 1912.

the University of Pennsylvania in 1874 and three years later received the degree of Doctor of Medicine from that institution. His industry during those student days foreshadowed the tireless energy of later years.

Following faithfully and with earnest purpose the scheduled exercises of the roster, he was alert to profit by any additional opportunities that might offer. Dissections abandoned by shirking classmates he promptly appropriated and carefully finished; the specimens of the autopsy table, neglected by too many, were to him Nature's page. Opportunities for practical laboratory and clinical instruction, which the official curriculum then denied, he secured by special and extramural courses. Among the group of students, who daily gathered after lunch to discuss the current work, young Musser soon won recognition as being not only well informed, but as possessing an exceptionally clear and analytical mind.

After completing his service as resident physician in the Philadelphia Hospital, the appointment, in 1879, as Medical Registrar in the University Hospital gave Dr. Musser additional opportunities for clinical and pathological study. That these opportunities were utilized is evidenced by his advancement, during the following year, to the responsible post of Chief of the Medical Dispensary in the University Hospital, which he filled from 1880 to 1886.

Following his election, in 1880, to membership in the Philadelphia Pathological Society, Dr. Musser became an active contributor to its *Proceedings*. It is interesting to note that his earliest communications related to pathological processes involving the circulatory organs, the stomach, and the liver—fields of internal medicine in which Dr. Musser later attained a wide reputation. His second paper, published in 1884, described a modification of the sphygmograph, thus indicating an appreciation of the value of scientific methods of clinical study.

The first ten years of his professional life were busy ones for the young physician, for during that period he was not only engaged in building up a rapidly growing private practice, but also in

zealously improving his opportunities to study disease. His appointments as Pathologist to the Presbyterian Hospital in 1884, Physician to the Philadelphia Hospital in 1885, and Physician to the Presbyterian Hospital in 1887 were recognitions of his ability, as well as important factors in his development.

Responsive to the opportunities of these positions and to the stimulus of association with some of the then leaders in American medicine, Dr. Musser became a frequent contributor to the journals and societies. His paper on "Cholecystotomy," written in collaboration with Dr. W. W. Keen, and read before this College, September 3, 1884, was an able plea for timely operation in biliary obstruction when such procedure was rare. The papers on "Idiopathic Anemia" and on "Primary Cancer of the Gall-Bladder and Bile-Ducts," among those which appeared during the next few years, bear testimony to Dr. Musser's industry and accurate observation.

To his activities as practitioner, pathologist, and hospital physician, those of teacher were early added. First as quizmaster in the Philadelphia Medical Institute—the arena in which so many distinguished teachers have won their first laurels—and then (1881) as Instructor in Clinical Medicine in the University of Pennsylvania, Dr. Musser demonstrated his ability to impart to others the results of his own diligent study. In 1889 he received the appointment of Assistant Professor of Clinical Medicine, which position he held in the University of Pennsylvania for ten years, until, in 1899, he became Professor of Clinical Medicine. This latter position he held at the time of his death.

Success in practice and a growing reputation as clinician and teacher paved the way toward his desire to devote his attention exclusively to internal medicine. After removing, in 1894, to the central part of the city, this ambition was realized, and during the last ten years of his life Dr. Musser's professional work was confined almost entirely to consultation.

His skill in diagnosis, the fruit of extensive observation and ripe judgment, but which at times seemed almost intuition, brought to his office and clinic cases from an extended territory.

His reputation as a diagnostician was so widespread that his opinion was sought by professional colleagues of many other and often distant cities. The hurried journeys, sometimes of a thousand miles or more, necessitated by these calls made frequent demands on his overcrowded days: from the sleeping car, with its night of broken rest, to his office and, after a hard day's work, back to the sleeper in response to some new summons was a not infrequent sequence.

Early appreciating the importance of more exact methods in clinical study, Dr. Musser was keenly alert to every advance in scientific medicine. The inmates of his hospital wards were sure of receiving the benefits of recent discoveries, if sanctioned by his own wise conservatism. The routine of his private consulting room showed the same regard for accurate methods, and while the demands of his daily obligations made personal attention to many of the deatils of clincial examination impossible, a well-organized laboratory, manned by competent assistants, formed an integral part of his equipment. The completeness of the records of his private cases shows an insistence on thoroughness and accuracy in daily routine.

An exceptionally strong constitution enabled Dr. Musser to indulge his indefatigable energy for many years without evident serious results. Just how much work he did accomplish only those intimately acquainted with his activities know. Busy days, filled with professional engagements until well toward midnight, were often prolonged into the small hours by literary labor, for many of his papers and most of the work on his *Medical Diagnosis* and book articles were written while others slept.

The earliest intimation that his health was seriously impaired was given in the autumn of 1910, when a slight attack of angina pectoris gave warning that habitual overwork was exacting its penalty. The emphatic verdict of colleagues at home and abroad, that remission of his professional work was imperative, was heeded and Dr. Musser sincerely promised himself a less strenuous program. While he did relinquish much of his work, he could not reconcile himself to a complete withdrawal from what had become

so much a part of his life. The knowledge of his precarious condition strongly influenced Dr. Musser in his decision to decline candidacy for the Professorship of Medicine in the University of Pennsylvania, when, in 1911, that chair became vacant.

Immediately following a meeting with his class at the University, Dr. Musser was seized with a severe attack of angina. His death occurred four days later, on April 3, 1912, and ended, when he was less than fifty-six years of age, a life so full of usefulness and high ambition. His often expressed wish, that he might fall in full armor, was realized.

As a practitioner, Dr. Musser brought to the bedside not only professional attainments of the highest order, but also a cheery humanness that administered an effective potion of confidence and hope. He was a firm believer in the value of medication, and his therapeutic resourcefulness was no small factor in his success. Ever alert to give those under his care the benefit of the latest advances in medicine, his ripe judgment and strict sense of obligation to the patient's welfare engendered an habitual conservatism. His sincerity, sympathy, and wise counsel won the loyalty of his extensive clientele to a remarkable degree, and his death was felt as the loss of a friend by those of humble and high station alike.

As a teacher, Dr. Musser, was eminently practical and direct. He possessed the happy faculty of being able to approach a subject from the level of the student, for his own diligent study and self-development had taught him to appreciate the difficulties and to anticipate the stumbling blocks that beset the student's path. He indulged in no flights of rhetoric or display of erudition, but in his simple and straightforward way, often emphasized by some homely but telling illustration, forced home the essentials of the art and science of medicine.

It was, perhaps, in his clinical conferences that Dr. Musser was at his best. His keen analyses of the cases, usually selected to exemplify some particular phase of disease, were convincing demonstrations of modern clinical methods, while his vast experience constantly supplied additional illustrations and suggestive

comments. He was anxious to repay his class with some new thought of practical significance, and took pains to instruct in the details as well as in the principles of treatment. The testimony of his pupils shows the high value set on these conferences, as giving so much of moment that was unobtainable from text-books.

Dr. Musser was continually considering and devising plans whereby the student might be brought into closer touch with actual phenomena, and every move to utilize more fully the hospital as the great clinical laboratory had his hearty support and coöperation. To his enthusiasm in this cause the University of Pennsylvania owes the establishment, in 1910, of its Department of Research Medicine, of which Dr. Musser was Director at the time of his death. The fulfilment of his dearest ambition, to win recognition as a distinguished clinical teacher, was signally achieved.

Dr. Musser's contributions to medical literature number over two hundred, and cover a wide range of topics. Of the ninety-odd technical papers which bear his name, more than a quarter relate to diseases of the stomach, the pancreas, and the biliary apparatus. It was, indeed, in this particular domain of internal medicine that his writings attracted attention abroad and won for their author an international reputation. Cardiac and pulmonary diseases also supplied the themes for other noteworthy contributions.

In response to many and repeated requests, Dr. Musser published, in 1894, his *Medical Diagnosis*, for the preparation of which his large experience and keen observation had so well fitted him. The book had a flattering reception and passed through five editions. He also contributed articles to several extensive works, among them Hare's *System of Practical Therapeutics* and Osler's *Modern Medicine;* edited the volume on *Diseases of the Lungs* in Nothnagel's *Encyclopedia*, and was, with Dr. A. O. J. Kelly, co-editor of a *System of Therapeutics*. In his writings as in his teaching, Dr. Musser was practical and direct, always emphasizing the value and importance of accurate methods in clinical study.

Although the increasing press of professional engagements necessarily limited active duty in public institutions, Dr. Musser continued to give valuable service to a number of hospitals and charitable foundations as consulting physician. At the time of his death, while visiting physician to only the University and Presbyterian Hospitals, he was consultant to the West Philadelphia Hospital for Women, the Philadelphia Hospital, the Rush Hospital, the Jewish Hospital, the Germantown Hospital, the Chestnut Hill Hospital, and the Woman's Hospital of Philadelphia.

As one of the physicians serving the interests of a great community, civic obligations claimed their share of his attention. He was active in seeking the betterment of Philadelphia's charities, and was deeply interested in the proposed work of the Vice Commission recently appointed by the Mayor of this city. Dr. Musser inaugurated the Tuberculosis Classes at the Presbyterian Hospital and actively aided in the Settlement Work of the University of Pennsylvania.

Realizing the futility of seeking to combat disease, when the patient's conditions of living and resources precluded effective treatment, Dr. Musser was deeply impressed with the importance of attempting to improve the hygienic and social conditions of many applicants at the Dispensary. His deep interest in this cause led to the establishment of the Social Service Work at the University Hospital, the first of its kind in this city. The splendid achievements of this service have amply justified the anticipations of its founder.

Dr. Musser served also as President of the American School Hygiene Association, Vice-President of the Pennsylvania Society for the Prevention of Tuberculosis, and was a member of the Board of Trustees of the Phipps Institute for the Study of Tuberculosis.

In addition to membership in the county, state, and national medical societies, Dr. Musser was a Fellow of the College of Physicians of Philadelphia, a member of the Association of American Physicians, the American Climatological Society, the

American Pediatric Society, the Philadelphia Pathological Society, and the Philadelphia Neurological Society. He also was a member of the American Association for the Advancement of Science, the Historical Society of Pennsylvania, and a member of the Board of Trustees of the Drexel Institute and of the Bureau of Municipal Research of this city.

In 1910 Dr. Musser received the honorary degree of Doctor of Laws from Franklin and Marshall College, while at the hands of his medical associates he had been honored by the presidency of the Philadelphia Pathological Society (1893-97), of the Philadelphia County Medical Society (1899), of the American Medical Association (1904), and of the National Medical Library Association (1910). He was an Honorary Member of the New York, Virginia, and South Carolina State Medical Associations and of the Academy of Medicine of Harrisburg.

Foreign recognition was not wanting, as he was elected Corresponding Member of the Sixth International Antituberculosis Congress (1907), Honorary Fellow of the Institute of Hygiene of London (1908), and Honorary Member of the Royal Society of Physicians of Buda-Pesth (1909).

Although his industry, enthusiasm, and ability in themselves would have insured success, the charm of Dr. Musser's personality was a potent adjunct in creating and maintaining the wide influence which he exercised. His was one of those rare natures whose magnetism attracted all who came within its spell, whether colleague or patient. The seriousness of earnest purpose was tempered by an abiding cheeriness so spontaneous and unaffected that his coming was welcomed by the strong no less than by the weak. The sombre shadows of the sick-room were riven by his smile, and even the grievously stricken often caught a ray of hope from his presence. Cordial and considerate toward all, his genial companionship made him everywhere a favorite guest. It was, however, those who enjoyed his intimate friendship who best knew the depth and loyalty of his frank and generous nature. Neither his enthusiasm for the best in his profession nor his exceptional ability in medicine, but his unswerving pursuit of

the highest ideals of living, it was, that won the deep and affectionate regard of his friends.

In 1879 Dr. Musser married Miss Agnes Gardner Harper. His wife and four children, three daughters and a son, survive him. It was with no little satisfaction that the father saw this son graduate in medicine and thus continue the traditions of the family by becoming the medical representative of the fifth generation.

The knowledge of his serious condition failed to disturb Dr. Musser's habitual cheerfulness, and few suspected that he was gravely ill. Realizing—and none more keenly than he—that he was mortally stricken, he never faltered, but pursued his daily rounds, relieving the suffering and brightening the lives of those whom he so faithfully served. Truly an impressive example of a steadfast courage born of absolute consecration to duty.

The concerns of the last days were never for himself, but only for those near and dear. And with the closing shadows came the peace and assurance that his years had not been lived in vain, that he had been true to ideals of high purpose, that the world had been the gainer for his being.

PRESENTATION OF A BUST OF GALEN

By RICHARD H. HARTE, M.D.

Mr. President and Fellows of the College:

It has been the time-honored custom of this Body, in presenting a portrait or statue to the College, to make some reference to the qualities of the individual as portrayed by his life.

The subject of this short sketch, Claudius Galen, was born in the year 131 of our era, and died at the age of seventy. He had the great advantage not always possessed, of being of gentle birth. His father was an architect, a man of rank and fortune and was distinguished both in letters and philosophy. He resided in Pergamos, in Asia Minor, where he bestowed on his son the greatest personal care regarding his education and gave him all the advantages that the world afforded at that time.

The young Galen, with a precocious intellect, availed himself of all the opportunities afforded in his native home and later by a sojourn in Rome, where he devoted himself rather to the study of the arts than to the science of medicine. There he acquired a broad foundation for his favorite study. This foundation he took with him to the then Medical Mecca—Alexandria—and there carefully studied the works of Hippocrates, for whom he had the greatest admiration and whose precepts he followed more closely than those of any other teacher. While at the school in Alexandria, he was an indefatigable seeker after all that was to be found in the field of medicine. Leaving Alexandria, he returned to his native city—Pergamos—where he for a time practised his profession.

Then, at the urgent request of the Emperor Marcus Aurelius, he came and settled in Rome and remained there for the greater part of his life. He was a person of great celebrity there, becom-

ing the Court physician. Aurelius referred to him, as Napoleon characterized Larrey, as the only upright man he knew.

Medicine in Rome in those days was evidently at a very low ebb, and the personnel of the profession (if such it might be called), was anything but pleasant, for a person of such culture and attainments as Galen to associate with. The so-called physicians —the great majority of them slaves—were thrown promiscuously together, where, in the spirit of the times, they waged war on each other, partly with unbridled language and partly with the poisoned methods of intrigue. They were all intent upon notoriety and regardless of the means by which it was attained.

In the midst of such environment, Galen lived and worked assiduously. But his success in Rome as a practitioner, investigator, and teacher soon found its counterpoise in many reverses and disconcerting experiences. It requires little imagination to picture the surroundings that existed in Rome at that time, particularly among so highly strung and excitable a community, where the physicians and their friends were the willing agents of any demagogues who chose to excite them to violence.

According to Galen's own account, fearing the worst from his enemies, whom he supposed to be planning his assassination, he left the capitol in the year 166. Probably his irascible temper, which he inherited from his mother, got the mastery of him at this time, and his leaving was largely the result of a fit of pique. However, this departure was before the advent of the great plague that devastated Rome and the surrounding country for fifteen years, having been introduced by the returning army from Smyrna. His traducers assert that Galen anticipated this epidemic and that it was a cowardly and unprofessional act to desert the capitol at this critical period.

After leaving Rome, he spent much time in travel and study in the East, only to be interrupted again by another summons from the Emperor to repair again to Rome. Galen returned with some reluctance, and once more found favor at Court, where his duties were not exacting. There he had ample time for congenial pursuits and he soon became one of the greatest

medical savants, eminent scholars, and most fruitful authors of all time. Many of his most valuable contributions to literature and science were written in Rome between the years 169 and 180.

Had we time, much might be said about Galen which would be most interesting, as his biographers have dealt very fully with him and his works. What interests us most tonight is the active part he played in the advancement of early medicine, many of the principles of which he established for all time.

His works on anatomy and physiology were marvelous, when we consider the times and facilities that were offered for study and investigation. Much of his anatomical study was done by dissecting animals and anthropoid apes. But in osteology, he was fortunate in procuring several skeletons which had been cleaned by the elements. His anatomical works were the best among the ancients, and continued to be used as text-books down to the sixteenth century. He was the first great experimental physiologist, as no one before his time habitually resorted to animal experimentation as a means of discovery in physiology.

Perhaps the discovery for which he is most noted is the true function of the arteries as vessels containing blood. He recognized the terminal communication between arteries and veins, and speaks of them as anastomoses. When we remember, that even in Harvey's time, this capillary connection between the arteries and veins had not been made visible by the microscope and that its absence was even thought a serious objection to his doctrine of the circulation, it seems a singular fact that it should have been taught by Galen 1500 years before. There is no doubt that Galen and others of his time practised the ligation of vessels for the arrest of hemorrhage; a method afterward lost; only to be revived by Paré in the sixteenth century.

His knowledge of practical surgery was most intimate and accurate. He speaks of wounds of vessels of moderate size being opened during an operation as one of the intercostals and suggests that the bleeding may be stopped by cutting the vessel completely across, as its divided ends retract and the orifices are closed.

His knowledge of the nervous system was as accurate and surprising as that of the circulatory system. In the pure field of medicine we find that he recognized malarial poisoning and speaks of the various types of this disease. Galen divided, for example, malarial fevers into quartans and tertians, and also recognized mixed and double infections, and states that a fever with attacks recurring every day is liable to be diagnosticated by the uninitiated as a quotidian. He wrote much about the pulse, on which he elaborated to a great extent but it was too academic to be of any practical value.

His description and treatment of pulmonary tuberculosis is practically as complete and accurate as our knowledge of the present day, excluding what we have learned by means of the microscope and tubercular bacilli. He thoroughly recognized the contagiousness of this disease.

Galen was the most versatile and voluminous writer of almost any age. Nearly two hundred treatises on the various branches of medicine have come down to us. Only the reverence in which his name was held saved his works from destruction when the great library in Alexandria was sacked and destroyed by the Alexandrians. The Christian vandals likewise preserved the works of Galen, though sparing few others of the ancient writings.

We have given but a short, fragmentary account of this remarkable man, who was undoubtedly a genius superimposed on a great man. The greatness Galen acquired was attained purely by the weight and power of his personality and the force of his solid achievement. It would seem as though he had lived before his time and the bright light which he shed on the field of medicine in his day was not destined long to shine, as, after his death, the medical world soon passed again into utter darkness, from which it but slowly recovered.

Mr. President, the bust which I take great pleasure in presenting to the College tonight is a faithful copy of one now in a private collection in Rome, and I have every reason to believe that it represents Galen in the possession of all the physical and mental vigor so characteristic of his active life.

THE ANNUAL ADDRESS OF THE PRESIDENT[1]

By G. E. de SCHWEINITZ, A.M., M.D.

One year ago a generous friend freed the College from its chief indebtedness, that it might, untrammelled, "pursue its career of honorable usefulness." This, I believe, it has done in the last twelve months, and that which follows is a record of what has been accomplished.

No special sessions of the College have taken place, but at the nine stated meetings, which have been held from December 1, 1910, to November 30, 1911, thirty-two papers were read, an increase of six as compared with the scientific programs of the same number of stated meetings of the preceding year. During this period of time the College listened to the Memoirs of three Fellows—namely, of Dr. J. Dutton Steele, prepared by Dr. John H. Musser and read by Dr. A. O. J. Kelly; of Dr. George C. Harlan, prepared and read by Dr. John B. Shober; and of Dr. De Forest Willard, prepared and read by Dr. Roland G. Curtin. It is not a little strange and more than sad that two of the readers of these Memoirs, Drs. Kelly and Shober, have died since that time, and of the work and activities of one of them the College has this evening heard; of the other it shall hear in the near future. All of the papers have commanded close attention, and in many instances elicited interesting discussions. Following a custom of previous years, it may be permitted to call attention to several of them of special interest:

[1] Read January 3, 1912

It was with no little satisfaction that the College realized that one of its former Presidents, Dr. H. C. Wood, had so far recovered from a long and tedious illness as to be able to send to the College an interesting communication on "Thoughts on the Teaching of Therapeutics Based on Forty Years' Experience," read for him, to quote his own words, by his most distinguished student, Dr. H. A. Hare. The Fellows listened with special satisfaction to the paper of Dr. George Fetterolf and Dr. George W. Norris on "The Anatomical Explanation of Paralysis of the Left Recurrent Laryngeal Nerve Found in Certain Reported Cases of Mitral Stenosis;" to the record of Dr. John B. Deaver's experience in the treatment of "Chronic Pancreatitis;" to "Further Clinical Studies in the Auscultation Method of Determining Blood Pressure," presented by Dr. Edward Goodman and Dr. A. Alexander Howell; to Professor Huber's well illustrated and scholarly paper on "Studies in Human Embryology;" and to Dr. Joseph Sailer's interesting account of the "Symptomatology and Treatment of Cecum Mobile." The beautiful exhibit of the transverse and sagittal sections of the adult head, prepared and demonstrated by Dr. Joseph Tunis, and the interesting cineomatograph exhibition of the gait, station, tremor, and general symptoms of nervous disease made by Dr. Weisenburg are worthy of special note. The paper on the quantitative analysis of human and of cows' milk recording the results of a long period of work by the late Dr. Arthur V. Meigs and by Dr. Marsh was followed by an instructive discussion from the chemical standpoint by Dr. John Marshall, and from the practical side by Dr. J. P. Crozer Griffith and Dr. E. E. Graham.

During these nine stated meetings 641 Fellows of the College were in attendance, an average attendance of $71\frac{2}{9}$, being an increase of 3 over that of last year and of 14 over that of two years ago. These figures do not, however, represent the actual attendance, because under the rules many physicians not members of the Fellowship were present who listened with satisfaction to the subjects that come up for discussion.

The Public Lectures on Great Doctors and Achievements in

Medical Research, to the pronounced success of which I referred in my last Address, continued in a delightful address by Dr. James G. Mumford, of Harvard University, on "Ambrose Paré and His Times," on the evening of April 18, 1911. On November 20th of the past year a second Public Lecture was given by Dr. Talcott Williams, of Philadelphia, not with reference to any one great doctor or his achievements in medical research, but descriptive of the doctor from the standpoint of the layman, a lecture which was at once quaintly humorous and brilliantly eloquent, and constituted a notable tribute to those who are banded together for the relief of human suffering and the prolongation of human life. This course will continue, and I can promise you that the next lecturer is sure to create a commanding interest.

In my last Address I announced the establishment of the Weir Mitchell Lectures and the selection of two lecturers, namely, the Professor of Pharmacology in the University of London, Arthur R. Cushny, and the Professor of Zoölogy in Columbia University, Edmund B. Wilson. Dr. Cushny discoursed on Auricular Fibrillation, and Dr. Wilson on Problems in Heredity, and their efforts were received with high appreciation by all who heard them. Following them came Dr. Svante Arrhenius, the distinguished Director of the Nobel Institute in Stockholm, who took for his subject "The Passage of Micro-organisms through Interstellar Space." His audience, composed of physicians, astronomers, and scientists generally, was keenly interested in his theories of the origin of life and in the experimental work which had led to their promulgation. An unusually large audience greeted the last Weir Mitchell lecturer of the year, Dr. William T. Porter, Professor of Comparative Physiology in Harvard University, who told the story of the cause and effects of Shock, and described his well-known work in this field of physiological research. To these lectures not only have the Fellows of the College and physicians in general been invited, but invitations have also been extended to senior students in the schools of medicine of this city and in

the scientific classes of neighboring colleges and universities, and the committee in charge of them has received many expressions of satisfaction that these unusual opportunities were thus freely offered.

The scientific business of the College during the past year has been excellent, and with my knowledge of papers that have been promised and my entire confidence in others that are to follow, I am sure that our record in the coming year will be equally good and doubtless better. In referring to this subject in a previous Address, I dwelt in words of high appreciation on the work of the Committee in charge of our Scientific Business, and particularly on that of its efficient Chairman, Dr. A. O. J. Kelly, whose untimely death, alas, has removed from our midst one of our best beloved and most active Fellows. The College is fortunate in that one has been found to take his place and to carry on the important work of this Committee with characteristic energy and excellence, Dr. Hobart Amory Hare.

Again, I desire to congratulate the Publication Committee on the material in the last volume of our TRANSACTIONS, and to emphasize a point made by the Chairman of this Committee, Dr. Davis, in his annual report, namely, that although Volume XXXII is not so large as the previous volume, because the latter includes the account of the Dedication exercises, it contains approximately one-third more of scientific matter. Once more I wish to impress upon the Fellows of the College the importance of these TRANSACTIONS, not only in that they enhance the scientific reputation of our institution, but because they are of great service in increasing our facilities for exchange with various foreign institutions of learning. I earnestly repeat the hope expressed last year that ways and means will be found to permit the publication in these TRANSACTIONS of lectures of special importance given in the College, and of valuable papers read before the Sections of the College.

Last year the Alvarenga Prize of the College of Physicians was won by one of Philadelphia's physicians, Dr. Francis D. Patterson, for his essay on "The Parathyroid Glandules." Since

its establishment in 1888 this prize has been awarded sixteen times, and among the successful competitors four have been Fellows of the College.

The Library. According to the report of the Chairman of the Library Committee, we know that at present the total number of volumes in the Library is 95,896, and that we possess 8917 unbound theses and dissertations, and 81,528 unbound pamphlets. From all sources during the past year the Library has received 4648 volumes, 14,791 pamphlets, and 26,074 numbers of various periodicals. The literary activities of our Fellows is evidenced by the fact that of the 660 new publications added to the Library during 1911, 46 were written or edited by members of our Fellowship. During the year, 1728 dissertations have been acquired, and the total number of periodical publications, American and foreign, during the same period of time is 1203, being an increase in subscriptions for the year 1911 of 59. The revision of the catalogue has made satisfactory progress, and the year has been a very successful one in the matter of exchanges, whereby, as the report of the Librarian shows, many valuable books and journals have been added to our collection.

It is again necessary to note that there has been a decrease in the number of visitors to the Library during the year, practically in the same proportion as in the previous report, which would indicate, as the Chairman of the Library Committee points out, that the change of location is still operative to a certain degree in this respect. This, however, is entirely offset by the fact that more than 16,000 books have been consulted, a very great increase in the number of those used in the previous year. This should be emphasized, inasmuch as it affords the best sort of evidence of the increased advantage which our library offers in its present quarters, and of the high regard in which it is held by those who most use it, and thus add to our scientific reputation and to the literary output of the medical profession of this city.

To another interesting statement in the report of the Chairman of the Library Committee I want briefly to refer, namely, to

the increased use of the private study rooms, the value of which was emphasized in my last Address, where it was pointed out that the number of volumes called for and retained in these rooms averaged about 50 the year around. In the year just ended this number has increased to 150. All visitors to the Library, and especially those who are accustomed to the difficulties of literary research, are greatly impressed with the value of these rooms, a value well demonstrated by the statement which has just been made.

The large increase in the number of accessions and number of bound volumes, as well as the decrease in the number of unbound theses and dissertations, is accounted for, as the Librarian and the Chairman of the Library Committee point out, by the fact that those in charge of the matter were enabled to bind more than 17,000 dissertations during the past year, adding in this manner 753 volumes to our shelves.

Last year I had the happiness to report, owing to the liberality of one Fellow and of one friend of the College, an increase of $6000 in the Library Endowment Funds. Unfortunately no such increase can be recorded during the past year, and I make reference to this matter in order most earnestly to appeal to the Fellows of the College to use their best efforts to bring this magnificent collection of books to the attention of those who have the means and willingness to give. No matter how great a library is, it cannot grow greater, or cannot continue to retain its greatness, unless it is adequately supported. We do splendidly with the money which we have, so judiciously spent by the Library Committee, but we need increased endowment. I pray you go out among those who can help us in this respect, and appeal for aid that we may become richer, and thus make those rich in knowledge and productiveness who come to read in our rooms.

Gifts to the College. The following is a list of rare medical books, works of special interest, paintings, engravings, and other valuable gifts received by the College during the past year.

Incunabula. (Total number, 160.)

Articella. Comentarii. Venetiis, Locatellus, 1493.
(Presented by Dr. J. H. Musser.)

Bartholomaeus de Pisis. Epitoma medicinæ. (Florentiæ, de Morgianis, circa 1490.)
(Fund for Rare Books.)

Matheolus Perusinus. De memoria augenda s.ars memorativa. Romæ, Plannck, (circa 1485-1490).
(Fund for Rare Books.)

Metlinger, B. Regiment der jungen Kinder. (Augsburg, Zainer, 1473.)
(Fund for Rare Books.)

Pintor, P. Aggregatior sententiarum doctorum de præservatione curationeque pestilentiæ. Romæ, Silber, 1499.
(Fund for Rare Books.)

Rhazes, A. Liber nonus, Venetiis, Papiensis, 1497.
(Fund for Rare Books.)

Roland of Parma. De curatione pestiferorum apostematum. Romæ, Udalricus Gallus, (circa 1471).
(Fund for Rare Books.)

Silvaticus, M. Liber pandectarum medicinæ. (Argentorati, Mentelin, circa 1470.)
(In exchange.)

Works of Special Interest.

Alexis of Piedmont. Secrets of that excellent physician. London, Wight, 1595.
(Fund for Rare Books.)

Blackmore, Sir R. Treatise upon the Small-pox. London, Clarke, 1723.

Chinese Punishments. Collection of water-colors illustrating forms of legal punishment in China.
(Presented by Dr. Thomas Biddle.)

Clinch, W. Historical Essay on the Rise . . . of the Small-pox. London, Warner, 1724.
(Purchased.)

Gesner, C. Nomenclator aquatilium animantium. Tiguri, Froschoverus, 1560.
(Presented by Dr. A. P. C. Ashhurst.)

Harvey, William. De motu cordis et sanguinis in animalibus. Patavii apud Sebastianum Sardum, 1643.
(Presented by Dr. S. Weir Mitchell.)

Martin, B. Essay on Visual Glasses. London, 1757.
(Presented by Dr. G. E. de Schweinitz.)

Memoranda of Dr. Benjamin Rush. (New York, 1910.) (Manuscript.)
(Presented by Dr. Robert Abbe.)

Russell, J. R. The History and Heroes of the Art of Medicine. 2 vols. London, Murray, 1861. (Extra-illustrated.)
(Presented by Dr. G. E. de Schweinitz.)

Spigelius, A. Opera quæ extant omnia. Amsterdami, Blæv, 1645.
(Presented by Dr. S. Weir Mitchell.)

(Withers, P.) History of the Royal Malady. London, 1789.
(Fund for Rare Books.)

Oil Paintings.

Portrait of Dr. William Goodell.
(Presented by the family of Dr. Goodell.)

Portrait of Dr. George C. Harlan.
(Presented by Mrs. Henry Post Mitchell.)

Portrait of Dr. Joseph Parrish.
(Presented by Mrs. Rodman Wharton.)

Portrait of Dr. George E. de Schweinitz.
(Presented by Mr. Rudulph Ellis.)

Portrait of Dr. James Tyson.
(Presented by his friends.)

Statues.

Marble statue of Æsculapius. (Heroic size.)
(Presented by Dr. Richard H. Harte.)

Medals.

Silver medal of Dr. Jacques Louis Reverdin by A. Carl Angst.
(Presented by Dr. W. W. Keen.)

Framed Pictures, etc.

Three photographs of the American Society of Clinical Surgery.
(Presented by Dr. Richard H. Harte.)

Engraving of James Benwell.
(Presented by Dr. George E. de Schweinitz.)
Photograph of Sir George A. Critchett.
(Presented by Dr. Walter L. Pyle.)
Engraving of bust of Dr. William Harvey.
(Presented by Dr. George E. de Schweinitz.)
Engraving of Dr. Benjamin Rush. Painted and engraved by Savage, 1800.
(Presented by Mrs. Gwendolen W. Howell.)
Photograph of Dr. De Forest Willard.
(Presented by Dr. De Forest P. Willard.)
Dedication of a book of poems to Dr. Nathaniel Chapman, 1838. (A poem by Dr. John Kearsley Mitchell.)
(Presented by Mrs. W. D. Winsor.)
Gold watch, formerly owned by Dr. Benjamin Rush.
(Presented by Dr. Robert Abbe.)

Our collection of incunabula continues to grow in size and importance, the total number in the College at the present time being 160, eight of these valuable volumes having been acquired by the College during the last year. With special pride we are able to point to our Harvey collection. From the statistics of the various editions of the works of Harvey which have been published, and which were gathered for a paper recently issued by Dr. S. Weir Mitchell, it is evident that in the various medical libraries in this country and in Europe there are forty-two editions. Of these forty-two our Library contains thirty-eight, including the first edition of "*de motu cordis*," 1628. Now only one other library, that of the Surgeon General's Office at Washington, contains as great a number, and in the near future we hope on some evening to make an exhibition of these works and to have the pleasure of listening to a discourse concerning them and him who wrote them from Dr. S. Weir Mitchell.

During the past year there have been added to our art gallery a portrait of Dr. William Goodell by his son-in-law, Mr. Alex. Scott, received through the kindness of his family; a portrait of Dr. George C. Harlan by Mr. Wallace V. Gilchrist, Jr., presented to us by Mrs. Henry Post Mitchell; a portrait of Dr. Joseph

Parrish, given by Mrs. Rodman Wharton; a portrait of the present presiding officer of the College by Mr. Julian Story, the gift of Mr. Rudulph Ellis; and a portrait of Dr. James Tyson by Mr. Joseph De Camp, which came to us from his many friends.

The formal presentation by Dr. Robert Abbe of the gold watch of Benjamin Rush took place on the evening of February 1, 1911. The College acts as the perpetual trustee of this valuable gift. The first custodian, Dr. Weir Mitchell, was appointed, and in the future the watch will be given from time to time into nominal custodianship of successive distinguished members of our profession.

The November, 1911, meeting of the College will always be memorable because on that occasion, at a charming and inspiring ceremony, the splendid statue of Æsculapius, a copy of one in the Vatican in Rome, the gift of the Treasurer of the College, Dr. Richard H. Harte, was presented by the donor in an admirable address rich in historical reminiscences and interesting legends, and received for the College by Dr. S. Weir Mitchell. It is unnecessary to elaborate the high appreciation of the College of this gift, which adds so much to the dignity and beauty of our building, and which commands the admiration of all those who enter it.

The Business of the College. The Fellows of the College have heard the satisfactory report of the Treasurer. To one or two points special reference may be made. The total assets of the College on December 1, 1911, were as follows:

Real estate, 13th and Locust Streets . . .		$150,000.00	
22d and Ludlow Streets			
Lot	$80,000.00		
Building	284,681.41		
		364,681.41	
Stable lot (less ground rent)		6,409.75	
			$521,091.16
Invested Trust Funds and Permanent Fund			180,304.98
			$701,396.14

The endowment of the Thomas Dent Mütter Fund for the Mütter Museum, about $30,000, and the Nathan Lewis Hatfield Prize Fund, about $7400, are not included in the above assets, as these funds are not in the hands of the Treasurer of the College. The actual income from all sources, including subscriptions, building fund, annual contributions, Hall Committee rentals, and Directory for Nurses for the year 1911, amounted to $42,104.73, while the expenditures during the year for all purposes amounted to $39,311.44, so that the actually unexpended balance to the credit of the College other than trust funds, permanent fund, building fund, and subscriptions, on December 1, 1911, was $3,114.56. On December 1, 1910, the Treasurer had a cash balance of $20,321.64, and on December 1, 1911, a cash balance of $23,114.56.

In my Address last year I recited in detail how, through the generosity of Mr. Eckley B. Coxe, Jr., the stable and three small dwelling houses adjoining the College on the south had passed into our possession, and referred to the permission that had been given that these small houses, because they were unsanitary, should be torn down. The removal of these houses was accomplished, and at the March meeting of the College the action of the President and the Treasurer in this respect was approved, and the Treasurer was further directed to raze to the ground the stable adjoining the College on the south, at the expiration of the present lease. This, as you know, has been done, and now only the lot once occupied by an unsightly building lies between us and our well-appointed neighbors upon the corner of Twenty-second and Chestnut Streets.

Because the Building Committee was able to announce that certain sums which had been promised to its fund had been paid, the College voted an appropriation of $1300 to be expended on improvements on the lot to which I have just referred. Therefore this matter has been taken into consideration by a committee composed of the President of the College, the Treasurer of the College, and the Chairman of the Hall Committee, who, in consultation with the architects, Messrs. Stewardson & Jamieson,

have studied a number of plans for beautifying this space of ground, and have the happiness to announce that the authorities of the Unitarian and Swedenborgian Church have entered heartily into all these arrangements, and will pay their share of the cost of the improvements.

Naturally, as long as the stable stood its rental reimbursed the College for its payment on the ground rent. With the expiration of the lease and the razing of the stable to the ground, this income, about $1500, ceased to exist, but the good fortune which has so constantly followed the College during the past few years and the generosity of the friends of the College which has so often been conspicuously evident have not failed us, and the Treasurer of the College has been able to announce that Mr. Eckley B. Coxe, Jr., has added to the deep debt of gratitude which we already owe him by the conveying this ground rent to the College, with the understanding that the College shall carry out a widely expressed intention to erect on this ground a suitable memorial to our late Vice-President and well-beloved Fellow, Dr. Wharton Sinkler. This condition I feel sure the College will gladly accept, and it will be only too happy to show its appreciation, not only of our late Vice-President, but of the great kindness of his nephew, by following to the best of its ability his wishes in this matter.

The Finance Committee has appropriated the necessary funds for the various departments of the College, and has endeavored with the money at its disposal to be as liberal as possible with these appropriations.

The Building Committee in consultation with the Hall Committee, has made one notable change in the Thomson room, which has proved to be eminently satisfactory, namely, the elimination of the dividing partition and the rearrangement of the chairs, so that they face the southern wall, against which the desk of the presiding officer rests, an arrangement which facilitates in every way the business of the meetings which take place in this room. The beautiful lamps decorating the posts of the great stairway are now in place, adding most gracefully to the light and decoration of our hall. With the aid of

an appropriation made by the College and certain donations from Fellows and friends of the College, an exceedingly valuable Leitz projection apparatus has been purchased by the Hall Committee, and is available for the use of the Fellows of the College and for the members of the scientific societies holding their meetings in our building.

The Hall Committee's satisfactory work is everywhere apparent. and it has been able to renew the contract for electric lighting at much more satisfactory figures than prevailed last year, Necessarily in the second year of occupation of a building of this character unusual expenses must arise, for example, in the renewal of paint and in repairing the general wear and tear. Therefore, it was necessary to grant the Hall Committee an increased appropriation. The Superintendent and the Clerk of the College have continued their effective work, to which special reference was made in my last Address.

The College has transacted all the routine business for the year, as well as all business brought to its attention from the Council, from the reports of special committees, and from resolutions presented on the floor of the College, save only that one committee appointed to select a design for a College gown has not yet made its report. One important and very satisfactory amendment to the ordinances and by-laws has been adopted, namely, the one defining the quorum at the meetings of the Council and giving to the Council certain limited executive functions, whereby some of the tediousness, or perhaps I should say the detail, of the business of the College is eliminated from the regular stated meetings, thus giving more time for the reading and discussion of scientific papers.

Concerning Those Who Have Died. During the year just ended seven Fellows have died.

Dr. John C. Da Costa, elected a Fellow in 1884, died on December 6, 1910. An accomplished surgeon, for many years gynecologist to the Jefferson Medical College Hospital, and at the time of his death emeritus gynecologist in that institution and consulting gynecologist to St. Agnes' Hospital, he gave liberally

of his skill and help to those who required his services, and added to the reputation of the special branch of surgery which he adopted as his life's work.

Dr. A. O. J. Kelly, elected a Fellow of the College in 1897, died on February 25, 1911. He was a wise physician, a keen clinician, an accomplished pathologist, an unusually effective teacher. A real student of the science, art, and practice of his profession, his literary productions had distinguished merit. He was a brave man, for he never faltered in his course nor shirked his work, albeit he knew only too well that for him the night was not far distant when no man can work.

Dr. Charles A. Oliver, elected a Fellow in 1884, died April 8, 1911. For many years he was an active and efficient member of the Library Committee of this College, and he has remembered in generous gifts the institution which he served. An accomplished ophthalmic surgeon, he devoted much time to the scientific side of his profession, and the results of his literary pursuits were rich in their fruits, giving him a well-deserved international reputation.

Dr. John B. Shober, elected a Fellow in 1891, died April 27, 1911. Devoting much of his time to gynecological practice, he had in recent years developed a deep interest in the therapeutic application of *x*-rays and of radium, and was in the best activity of his researches on the use of these agents which promised notable results when his career was checked by his untimely death.

Dr. Michael T. Prendergast, elected a Fellow in 1905, died June 3, 1911. For many years attending physician on the staff of St. Joseph's Hospital, he conducted a large and successful practice, was noted for his devotion to his work and for the high regard in which he was held by his fellow practitioners and by all of those who came in contact with him.

Dr. Joseph Price, elected a Fellow in 1889, died June 6, 1911. To him many came for instruction, for his skill was noted and his reputation widespread, and to him some of the foremost gynecologists of this country owe their earliest training in this branch

of surgery. He was vigorous, often aggressive, but not always wise in the expression of his opinions, saying that which he thought was right, asking no favors and being unafraid. He was a notable and interesting personality, who worked until his last hour, and died in his harness.

Dr. Edward Shippen, elected a Fellow in 1876, died June 16, 1911. He was a valiant servant of his country, rendering to her distinguished aid in the trying days of the Civil War as Surgeon in the Navy and later as Medical Director of this branch of the service. At the time of his death he was Rear Admiral of the United States Navy on the retired list.

Two Associate Fellows died during the year: Dr. Edward G. Janeway of New York, in the foremost rank of the great clinicians and consultants of this country, died February 10, 1911. Dr. Henry P. Bowditch of Boston, a distinguished teacher and a physiologist of enduring reputation, died March 13, 1911.

Thus the College has lost by death during the year, as it did in the preceding year, seven Fellows. The resignation of four Fellows has been accepted. Sixteen new Fellows have been elected. The net gain, therefore, is five. On December 1, 1911, the roll of the College was as follows: Fellows, 456; Associate Fellows (American), 25; Associate Fellows (foreign), 19; Corresponding Members, 4; a total of 504.

In an earlier portion of this Address I bespoke your interest in the endeavor to increase the endowment of the Library, and have urged that efforts be made to find means and ways to include in our TRANSACTIONS important papers now excluded because of lack of funds. I beg of you to take these matters into serious consideration.

While it is necessary and important that the Fellows should be familiar with the business of the College recorded in the reports read by the Treasurer, the Secretary, and the Chairmen of the eight Standing Committees at the December meeting of each year, it must be apparent to all of us that the technique of the presentation of these essential matters is subject to improvement.

Two plans suggest themselves: (1) That these reports be printed, and copies of them be distributed to the Fellows, and that at the December meeting the author of each Report be allowed a few minutes, five at the utmost, in which to emphasize any statements he may desire to bring to the especial attention of the members. This plan of presenting these reports, especially that of the Treasurer, has on more than one occasion been proposed, especially by my predecessor. It would materially shorten an otherwise necessarily long session, and give to the Fellows a familiarity with the business of the College which few of them now possess. Its chief objections would seem to be that it would entail a certain amount of expense, and that there is doubt whether the copy of these reports could be prepared in time for printing before December 1 of each year. (2) That a special meeting of the College be called, during the first week of each December, for the sole purpose of listening to these reports read as is now the custom. The chief objection to this plan is that it would be difficult to assemble an audience. The plan of printing and distributing the reports, adopted by many societies, associations, and clubs, seems to me the better one, but I would suggest that the College authorize the appointment of a committee to take the matter into consideration and to report the results of its deliberations.

The Fellows are aware that in earlier days the College frequently concerned itself, sometimes on its own initiative and often by request, with matters not restricted to the presentation of scientific papers, that the College, in the language of Dr. Tyson, was actively interested in the health and even morals of the community. In more recent times this interest has declined, doubtless, as the Honorary Librarian points out, because of the rise of influence in these respects of the County and State Societies, and the College has devoted itself more exclusively to its scientific programs and to the elaboration of its Library, although, as its minutes and its actions well show, it has by no means entirely abandoned this field of activity. Be that as it may, it has been in the minds of some of the Fellows that this

work should be rejuvenated, and one of our membership, Dr. James M. Anders, was prepared to present a paper on this subject and open it up for discussion. Unfortunately, for lack of time at the meeting at which this paper was listed, it was necessary to postpone it to a session in the near future, but by permission of Dr. Anders I am now calling attention to this matter in order that the Fellows may be prepared to receive with intelligent interest the suggestions he will make. He will urge that the public lectures shall include from time to time such subjects as the importance of hygiene, sanitary science, and preventive medicine, presented in such a manner that they would interest the public in carrying on a campaign of education and would furnish practical information on health topics. He will further urge the establishment in the College of a standing committee on hygiene, sanitation, and preventive medicine. My own opinion on this subject would be out of place until the matter comes up for discussion on the occasion of the reading of Dr. Anders' paper, to which I am sure the Fellows will listen with keen interest. I make only this reference to it for the reason already stated.

To our incunabula and our Harveys I have referred, as well as to the contemplated exhibition of the latter collection and to Dr. Mitchell's paper concerning it. But we have a large number of ancient and rare medical books other than those I have named. The time is ripe for calling especial attention to them, and I venture to suggest to the Section on Medical History that an evening be selected when these books can be suitably displayed, the chief among them briefly described, and that to this function shall be invited not only the Fellows, who, indeed, would come by right, but the officers of the Library Companies of this and neighboring cities, and all those who are interested in, and have a love for, ancient literature. It seems to me that such a night would afford a rare enjoyment.

In this month, 125 years ago, our College was formally organized, John Redman presided, the constitution was read, approved, and ordered to be printed, and in its later amended form has been signed by every Fellow who has been elected since that day.

On this roll on the first day of last December 935 names had been inscribed. It is a precious, safely guarded volume; between its leaves lurk the shadows of a century and a quarter. In his inaugural address the first President of this Collegiate Body prayed that the Fellows who heard him and that those who were to follow in their footsteps would with wisdom, prudence, discretion, and judgment publicly and privately serve their day and generation. This they have done, and I hope that in the very near future we may in some formal manner celebrate this, on which I extend to you hearty congratulations, our one hundred and twenty-fifth birthday.

THE ANATOMICAL EXPLANATION OF THE RELATIVELY LESS RESONANT, HIGHER PITCHED, VESICULOTYMPANITIC PERCUSSION NOTE NORMALLY FOUND AT THE RIGHT PULMONARY APEX[1]

By GEORGE FETTEROLF, M.D., Sc.D.

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AND

GEORGE WILLIAM NORRIS, A.B., M.D.

ASSISTANT PROFESSOR OF MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA; VISITING PHYSICIAN TO THE EPISCOPAL HOSPITAL AND TO THE OUT-PATIENT DEPARTMENT OF THE PENNSYLVANIA HOSPITAL; ASSISTANT VISITING PHYSICIAN TO THE UNIVERSITY HOSPITAL; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

(From the Laboratory of Anatomy, University of Pennsylvania)

Most clinicians, especially those who have devoted particular attention to diseases of the lungs, are agreed that in percussing the upper parts of the chest a slight difference in sound is normally heard when the two sides are compared. This difference consists in the fact that on the right side, from the apex down to about the level of the second interspace or the third rib, the note elicited is slightly higher in pitch, shorter in duration, and less resonant. Occasionally, also, the note is somewhat more tympanitic in quality.

The actual amount of difference in the afore-mentioned sound attributes, while never great in degree, is of extreme clinical importance on account of the frequency with which incipient tuberculosis manifests itself by physical signs at the right apex. If no allowance be made for a normal variation in the percussion sounds of the two sides of the chest, errors in diagnosis easily may occur.

[1] Read January 3, 1912.

On consulting various text-books on the subject one finds that in reference to the question under consideration they may be divided into four classes: (1) Those in which allusion is made to the difference, but no explanation is offered. (2) Those in which no mention of any difference is made. (3) Those in which it is denied that any difference exists. (4) Those in which more or less explanation of the phenomena is attempted.

The following statements are taken from works on physical diagnosis which comprise the first class:

"On the right side the sound is somewhat less clear, shorter, and of a higher pitch than on the left." (J. M. DaCosta.[1])

"The pulmonary quality is slightly less marked on the right than on the left in supraclavicular and infraclavicular areas." (G. R. Butler.[2])

"It [the resonance] is slightly higher in pitch at the right than at the left apex." (J. H. Musser.[3])

Similar statements are made by J. C. Wilson[4] and R. C. Cabot.[5]

Among the authors who make no mention of the normal variation in the percussion note of the two sides are: Loomis,[6] Gibson and Russell,[7] Vierordt,[8] J. H. Bass,[9] P. Simon,[10] S. Gee,[11] A. Weil,[12] P. Niemeyer,[13] Ebstein,[14] Stokes,[15] Sahli,[16] Hare,[17] Fowler and Godlee,[18] and Fuller.[19]

In the third class we found only one book, that by S. G. Bonney,[20]

[1] Medical Diagnosis, 1900, p. 270.
[2] The Diagnostics of Internal Medicine, p. 426.
[3] A Practical Treatise on Medical Diagnosis, 1899, p. 495.
[4] Medical Diagnosis, 1909, p. 131.
[5] Physical Diagnosis, 1909, p. 130.
[6] Lessons in Physical Diagnosis, 1887, p. 27.
[7] Physical Diagnosis, 1902, p. 142.
[8] Lehrbuch d. klin. Untersuchungsmethoden, 1904, i, 112 and 634.
[9] Medizinische Diagnostik, 1883, p. 112.
[10] Man. de Percussion et d'Auscultation, 1895, p. 35.
[11] Auscultation and Percussion, 1907.
[12] Handbuch u. Atlas der Topograph, Perkussion, 1877, p. 42.
[13] Handbuch d. theor. u. klin. Perkussion u. Auskultation, 1870, p. 130.
[14] Leitfäden d. Aerztlichen Untersuchung, etc., 1907, pp. 151 to 158.
[15] Diagnosis and Treatment of the Diseases of the Chest, 1837, Part 1.
[16] A Treatise on Diagnostic Methods of Examination, 1905.
[17] Practical Diagnosis, 1906.
[18] Diseases of the Lungs, 1898.
[19] Diseases of the Lungs and Air Passages, 1867, p. 51.
[20] Pulmonary Tuberculosis, Philadelphia, 1908, p. 183.

who says: "The characteristics of normal resonance are identical upon the two sides of the chest in this (infraclavicular) region, as contrasted with the striking differences displayed below the third ribs."

The text-books of the fourth group offer numerous explanations of the phenomena.

The explanation is based on the thickness of the pectoral muscles by the following writers: A. Flint,[1] J. G. Brown,[2] Edlefsen,[3] Beevor,[4] Guttman,[5] E. Cassaët.[6]

Eichhorst[7] says that the difference is most marked in men of laborious occupations. He goes on to say, however: "Bei Linkshaendingen habe ich den Perkussionsschall beiderseits laut gefunden."

Brugsch and Schittenhelm[8] and C. L. Greene,[9] regard the difference as due to the nearness of the large bronchus.

Kidd[10] attributes the variation in part to the difference in the bronchi, stating that the right main bronchus is slightly wider and more vertical, and that the bronchus to the upper lobe is given off higher up, that is, near to the trachea.

In answer to this it may be stated that the point at which the upper lobe bronchus of the right side is given off is opposite the upper border of the third costal cartilage. Its effect on resonance at the apex must be very slight.

Walshe[11] states that: "In a certain number of persons whose lungs are perfectly sound the right infraclavicular region gives a more wooden note under percussion, and is at the same time less resonant than the left. This peculiarity is perfectly unconnected with excess of muscular substance on the right side, and depends on some hitherto unascertained condition."

[1] Variations of Pitch in Percussion and Respiratory Sounds, 1852, p. 9.
[2] Medical Diagnosis, Edinburgh, 1887.
[3] Lehrbuch d. Diag. d. inn. Krankheiten, 1889, p. 449.
[4] Lancet, May 4, 1901, p. 1275.
[5] Lehrbuch der Klin. Untersuchungsmethoden (Klemperer), 1904, p. 145.
[6] Precis d'Auscultation et de Percussion, 1906, p. 563.
[7] Lehrbuch d. Physikal. Untersuchungsmethoden, Berlin, 1889, i, 269.
[8] Lehrbuch Klinischer Untersuchungsmethoden, 1908, p. 80.
[9] Medical Diagnosis, 1910, p. 101.
[10] Allbutt's System of Medicine, 1909, v, 346.
[11] Practical Treatise on the Diseases of the Lungs, 1871, p. 64.

In another place[1] the same author writes that: "Numerous differences in the results of percussion of corresponding points of the two sides of the chest have been noticed, and traced to an obvious cause the presence of texture and organs of different densities in those spots. Variable thickness of the external soft parts will have a similar effect; thus the right infraclavicular region is less resonant than the left in robust persons whose employment requires much use of the right arm; the pectoralis muscle enlarges from use. Here the explanation is obvious, but the explanation is not obvious when the right infraclavicular region emits less tone than its fellow, in persons presenting no muscular thickening of this kind. Such inferiority of resonance (sometimes attended, too, with slight elevation of pitch) is never great provided the lungs are perfectly healthy; it holds good whatever be the direction given to the fingers, and whether they fall outward toward the humerus or inward toward the sternum."

H. S. Anders[2] attempts an explanation by stating that in addition to greater thickness of the pectoral muscle and the presence of the liver on the right side: "An explicit reason is found in the fact that there is a different anatomical arrangement of the bronchial tubes, those on the right side being larger and situated more superficially and higher up; this, with a bunch of medium and small branches occupying space, that on the left side is filled with air vesicles, gives an amount of bronchovesicular tissue sufficient to elevate the pitch slightly but perceptibly, the tubes adding an element of tympany, the extra muscular and connective tissue of their firm walls an element of dulness."

Anders and Boston[3] state: "The higher pitched percussion note over the right than over the left apex is probably due to larger diameter and higher position of the right bronchus. The note is again modified by the thickness and tension of the chest wall as the result of muscular contraction," etc.

Gerhardt[4] offers the following reasons: (1) Increased size of the

[1] Lancet, 1849, i, 196.
[2] Physical Diagnosis, 1907, p. 99.
[3] Text-book of Medical Diagnosis, 1911, p. 55.
[4] Krause's Lehrbuch d. klin. Diagnostik innerer Krankheiten, 1909, p. 122

muscles on the right; (2) slight scoliosis often present; (3) difference in anatomical structure: (*a*) bronchi larger, (*b*) pulmonary apex smaller and lower, (*c*) bronchi retain large caliber near to the surface.

Inasmuch as none of these explanations are adequate to account for all cases, we have endeavored to solve the problem by the careful anatomical study of frozen sections of the cadaver, previously hardened in formalin, and by a critical study of the chests of 32 left-handed students. The examinations of the left-handed individuals were made with especial reference to the following points: (1) Circumference of the chest, right and left. A tape-measure was applied to the side of the chest, extending from the middle of the spine to the midsternum at the level of the nipple. For obvious reasons errors in such measurements may easily be made, and we, therefore, lay no great stress on the importance of the data thus obtained. The results show, however, that the *right* chest is not infrequently larger even in left-handed persons. (2) Vocal fremitus, elicited below the clavicle. (3) Percussion resonance, from the clavicle to the upper border of the third rib. (4) Percussion pitch, elicited over the same area. (5) Character of the breath sounds in the same region. Corresponding points on the two sides of the chest were of course compared, and the results immediately recorded.

The accompanying table represents in charted form the results of our examinations, and the data obtained were so uniform that it seemed needless to examine a larger number of men. From these studies the following conclusions may be drawn:

Vocal fremitus is normally more intense on the right side, even in left-handed individuals, and probably is due, as was pointed out by one of us (Fetterolf[1]), to the immediate contact of the trachea to the right pulmonary apex. On the left side the aorta, the common carotid and subclavian arteries, and the esophagus, together with lymphatic and areolar tissue, a layer some 2 or 3 cm. in thickness, are interposed between the lung and the trachea.

[1] Arch. Int. Med., February, 1909.

TABLE OF RESULTS OBTAINED BY EXAMINING THE CHESTS OF THIRTY-TWO LEFT-HANDED INDIVIDUALS

No.	Circumference of chest (inches)		Vocal fremitus		Percussion resonance		Pitch (higher)		Breath sounds (harsher)		Remarks
	R	L	R	L	R	L	R	L	R	L	
1	17¼	17	+	..	−	..	+	..	Equal		
2	14½	14	+	..	−	..	+	..	Equal		Poorly developed; 18 yrs. old; looks to be 14.
3	16¼	16	+	..	−	..	+	..	+	..	
4	18¾	18	+	..	−	..	+	..	+	..	Large deep pectoral muscles; difference only on deep percussion.
5	16	15¼	+	..	−	..	+	..	+	..	Large left pectoral muscles; difference slight.
6	16½	16½	+	..	−	..	+	..	+	..	Well-marked difference.
7	17½	17¾	+	..	−	..	+	..	+	..	
8	16½	16¾	+	..	−	..	+	..	+	..	
9	15¾	16¼	+	..	−	..	+	..	+	..	
10	16	16	+	..	−	..	+	..	+	..	Bilateral subclavian murmur.
11	15½	15¾	+	..	−	..	+	..	+	..	Difference slight. Funnel breasted.
12	17¾	17½	+	..	−	..	+	..	+	..	
13	15½	16	+	..	−	..	+	..	+	..	Marked retraction of whole right side.
14	16½	16½	+	..	−	..	+	..	+	..	
15	17¾	17½	+	..	−	..	+	..	+	..	
16	16	16	+	..	−	..	+	..	+	..	
17	18	17	+	..	−	..	+	..	+	..	
18	17	16½	+	..	−	..	+	..	+	..	
19	16½	17½	+	..	−	..	+	..	+	..	
20	17¾	18¼	+	..	−	..	Equal		+	..	
21	16½	18	+	..	+	..	+	..	−	+	Flat left chest; looks pathological.
22	18¼	18½	+	..	−	..	+	..	+	..	
23	15½	15¼	+	..	..	+	..	+	Equal		Very poor expansion; feeble breath sounds.
24	18½	18½	+	..	−	..	+	..	Equal		
25	18¾	18	+	..	−	..	+	..	+	..	
26	17	17	+	..	−	..	+	..	+	..	
27	16½	17	+	..	−	..	+	..	+	..	
28	15¾	15¾	+	..	−	..	+	..	+	..	
29	16	15¾	+	..	−	..	+	..	+	..	Difference reversed on light percussion.
30	18½	18	+	..	−	..	+	..	+	..	
31	18	17½	+	..	−	..	+	..	+	..	
32	16¾	16¾	+	..	−	..	+	..	+	..	

While studying the percussion sounds at the two apices it was occasionally noted that after prolonged percussion differences quite noticeable at first tended to become less marked. This is by no means an original observation, and may be explained as due to one or all of the following factors: (1) Aural fatigue on the part of the examiner. (2) A pulmonary reflex, as described by Abrahms, by virtue of which percussion over a certain area of lung tissue produces a local reflex dilatation of the air vesicles, thus obscuring a slight degree of dulness. (3) Spasm of the deeper fibers of the pectoral muscle, analogous to the increased myotatic irritability of myxedema seen in low nerve tone,[1] which by increased muscular density tends to limit the penetration of the percussion vibrations.

In persons of poor muscular development, in case of the aged, the wasted, and the muscularly inactive, the difference in the pulmonary resonance is present, while a corresponding unevenness in the thickness of the pectoral muscles may be either inconsequential or entirely lacking. The main cause, then, must lie deeper than in the muscles of the chest.

Let us consider for a moment what physical conditions underlie the changes in the percussion note which we are endeavoring to explain, namely: (1) diminished resonance; (2) elevated pitch; (3) a tympanitic element.

1. *Diminished resonance* over normal lung implies a lessened quantity of air and elasticity or the presence of overlying tissue, which by reason of its density vibrates less complexly, and transmits vibrations less well. The result, acoustically speaking, is a condition in which the fundamental note preponderates and fewer overtones are heard. The sound is more muffled and is shorter and weaker, tone enforcement being diminished. With the appearance of dulness there is always an increase in the elevation of pitch.

2. *Elevation in pitch* implies an increased vibratory rate, and this in normal pulmonary tissue again depends largely, other things being equal, on the amount of air, the size of the air columns.

3. *A tympanitic quality* is heard over pulmonary tissue when:

[1] This explanation, used in a somewhat similar connection, has been suggested by M. E. Rist, Percussion Thoracique, La Presse Médicale, December 16, 1911.

(*a*) Air columns vibrate in smooth walled chambers which communicate with the atmosphere; (*b*) when air in closed cavities vibrates in unison with elastic walls; (*c*) when the normal pulmonary tension is relaxed, so that the lung vibrates as a whole (for example, in pleural effusion). Acoustically the underlying characteristic of tympany is preponderance of overtones. For the present purposes only the first proposition (*a*) need be considered.

It may be stated as a general principle that a less resonant note is produced by an increase in the amount of airless tissue. Writers who have hitherto considered this to be a factor have confined their explanation to conditions of the chest wall, assuming that right-handed people have so much more muscular tissue over the pulmonary apex on that side that the percussion note becomes dulled in quality. Thus two left-handed individuals were exhibited before the Clinical Society of London by Sir Hugh Beevor to prove this very point, that is, that the left side was less resonant.[1] Similar conclusions were reached by Brazil,[2] who, having studied a number of cases, states that the higher pitched note on the left in left-handed people affects the entire chest, and is not limited to the apices. How this can be it is difficult to understand, because in the axillary region, for example, muscular development plays practically no role.

Similar views are held by E. Lloyd Jones,[3] who in an interesting article quotes as corroborative evidence the fact that in a right-handed patient of his the left side was less resonant than the right on account of a "former venous thrombosis in the left arm," the veins in the upper part of the left chest being much enlarged. It seems to us that Jones' explanation, which links a diminished resonance with the presence of thrombotic vascular changes on the left side, is in entire consonance with the explanation proposed in this communication. We believe, however, that the percussion changes in such a case as he reports are due more to a distention of the large vessels crossing the apex than to a thickening of the superficial tissues.

[1] Lancet, May 4, 1901, p. 1275.
[2] British Med. Jour., October 26, 1899, p. 920.
[3] Ibid., October 24, 1903.

AUTHORS' EXPLANATION.

As the result of our studies we believe that the anatomical basis for the conditions under discussion consists of three factors, namely, the anterior position of the vessels in relation to the right apex as compared with the left, the consequent encroachment upon and reduction in size of the right apex, and the apposition of the trachea to the inner surface of the right apex, while the same aspect of the left is in contact with a thick layer of solid tissue.

FIG. 1.—Mediastinal surface of lungs hardened before removal. This photograph shows (*a*) the groove produced in the right apex by the trachea; (*b*) the relatively anterior position of the vessels on the right side; and (*c*) the smaller size of the right apex. *AG*, azygos groove, *VG*, grooves for superior vena cava, innominate vein, and subclavian vessels; *TG*, tracheal groove; *SG*, subclavian groove; *AAG*, aortic groove; *CI*, cardiac impression.

Reduced to their last analysis the anatomical differences between the two sides in the upper thorax will be found to be due to the fact that man is a sinistro-aortic individual. In some of the lower vertebrates, for example, the birds, the aorta is on the right side, and in others, as some of the lizards, there is an aorta on both sides. In the early human embryo, when the heart is situated far forward in the pharyngeal region, the arrangement of the large arteries is

symmetrical. As development proceeds the main trunk, the aorta, is established on the left side.

In its course from the heart to the vertebral column the systemic aorta passes first slightly to the right, then slightly to the left, and finally almost directly backward between the left side of the trachea and the mediastinal aspect of the left lung. Thus situated it affects both the trachea and the lung, throwing the lower end of the former over toward the right, thereby producing the now well recognized dextro-inclination of the trachea, and grooving deeply, in an arciform manner, the inner aspect of the left lung, in front, above, and behind the root of the latter.

FIG. 2.—Anterior view of lungs hardened before removal. This photograph shows the anterior position of the groove for the subclavian vessels on the right side, compared with the more superior position on the left. *SG*, subclavian groove.

The aortic arch is usually divided by writers into three portions, an ascending, a transverse, and a descending. The first and third of these parts are properly named, but "transverse" is incorrectly applied to the second portion. As regards the aorta, it should be abolished from anatomical terminology and some such term as "horizontal" substituted for it. Its use in this connection probably originated from the fact that when the chest is opened under ordinary condition, the collapsed lungs and the heart fall backward,

and the second portion of the arch becomes approximately transverse. But when the thoracic contents are hardened before the chest is opened it is seen clearly that there is no justification for applying to the second part the term "transverse." When examined under such conditions the general course of the aortic arch is seen distinctly to be almost anteroposterior, there being, of course, a slight tendency to run toward the left. This is demonstrated most clearly in all of the sections that we have prepared; and in nearly

FIG. 2.—Lateral view of lungs hardened before removal. This photograph shows the deeper vascular groove and the smaller size of the right apex as compared with the left. *SG*, subclavian groove.

all of those cut in the sagittal plane the entire aortic arch from beginning to end is included in the section.

As stated above, we believe that there are three ultimate factors at work to produce the normal discrepancies at the apices.

First. As regards the anterior position of the vessels of the right side. The first branch, better called the anterior branch, of the horizontal part of the aortic arch is the innominate artery, destined for the supply of the right head and the right upper extremity.

Arising, as it does, to the left of the median line, it is compelled to pass anterior to the trachea in order to reach its destination. This position of its origin is responsible for the anterior situation of the vessels in relation to the right apex. Having crossed the anterior aspect of the trachea and turned somewhat posteriorly, it divides

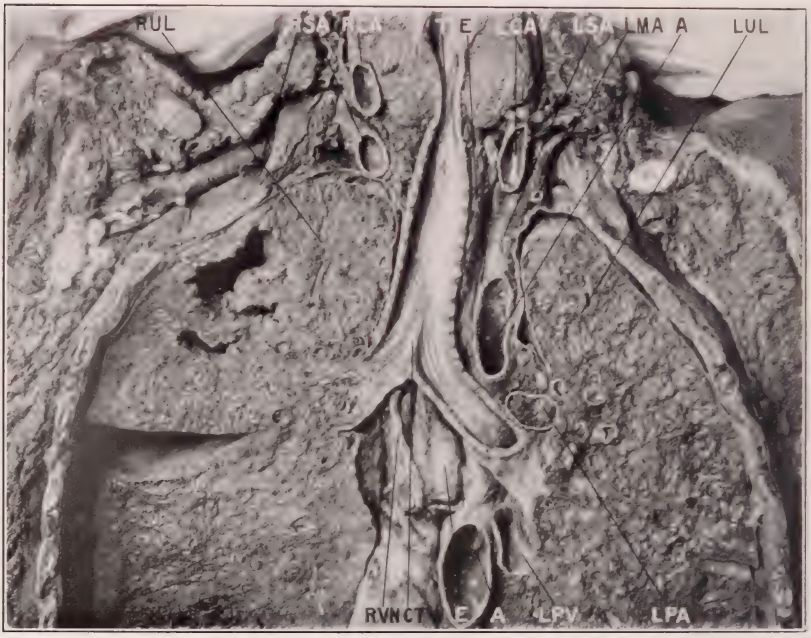

FIG. 4.—Frontal section of the thorax in the axis of the trachea This photograph shows (*a*) the contact of the right upper lobe with the trachea, (*b*) the mesial position of the left subclavian artery in relation to the left upper lobe, and (*c*) the essentially anterior position of the right subclavian in relation to the right upper lobe. *RUL*, right upper lobe; *RSA*, right subclavian artery; *RCA*, right common carotid artery; *T*, trachea; *E*, esophagus; *LCA*, left common carotid artery; *LSA*, left subclavian artery; *LMA*, left internal mammary artery; *A*, aorta; *LUL*, left upper lobe; *LPA*, left pulmonary artery; *LPV*, left inferior pulmonary vein; *CT*, carina tracheæ; *RVN*, right vagus nerve. (Reproduced from the Archives of Internal Medicine, February, 1909.)

into its terminal branches, the right common carotid and the right subclavian, the former of which does not concern us. The latter passes obliquely upward and *backward* to reach its groove on the upper surface of the first rib. In its course it produces a deep sulcus in the anterior aspect of the pulmonary apex.

The second branch of the horizontal part of the arch is the left common carotid. It passes upward at the side of the trachea, between the trachea and the left apex, and enters the neck.

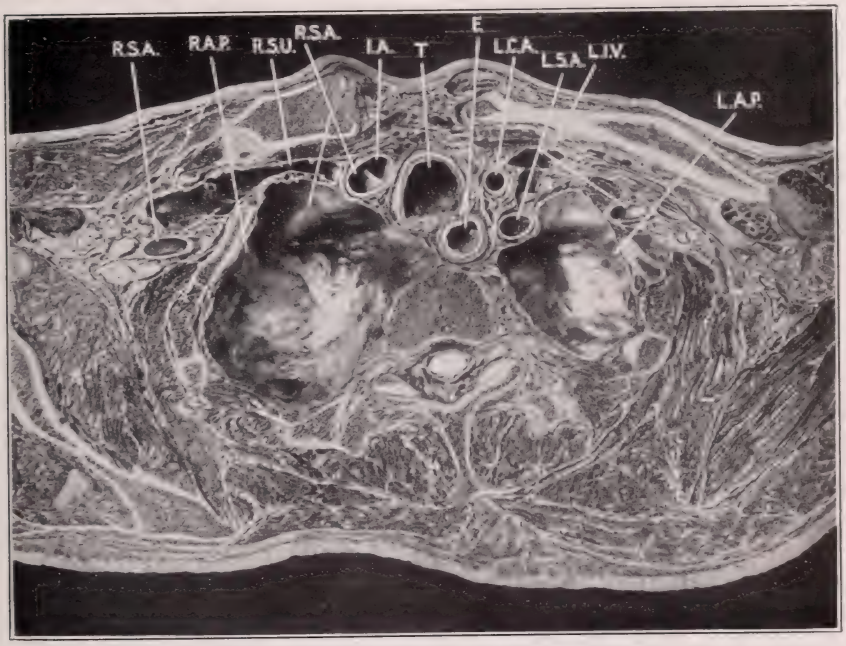

FIG. 5—Section through the upper part of the thorax, viewed from below. The line of section is not exactly horizontal, a slightly lower plane being reached on the right side than on the left. In order to show the apical parietal pleura, the pulmonary apices have been removed. There can be noted in this specimen the beginning contact of the right pleura with the trachea, and the anterior position of the innominate artery, whose bifurcation is well shown. On the left side, the wide separation of the pleura from the trachea by means of the large arteries, esophagus, and areolar tissue can readily be seen. There can be seen well the deep position of origin and the obliquely anterior course of the left subclavian artery. *T*, trachea; *E*, esophagus; *RAP*, right apical pleura; *LAP*, left apical pleura; *IA*, innominate artery, dividing into *RSA*, right subclavian artery and *RCA*, right common carotid artery; *LSA*, left subclavian artery; *LCA*, left common carotid artery; *RSV*, right subclavian vein; *LIV*, left innominate vein.

The third or posterior branch of the horizontal part of the arch is the left subclavian. This vessel is given off well back in the mediastinum, opposite the posterior surface of the trachea, or even at the left side of the esophagus. It arises so deeply that it is compelled to pass slightly *forward* as well as laterally to reach its groove on the first rib. In the greater part of its thoracic course it occupies

the layer of tissue between the left upper lobe and the trachea. Nearing the first rib it produces in the left apex a groove which is shallower and nearer the summit than on the right side.

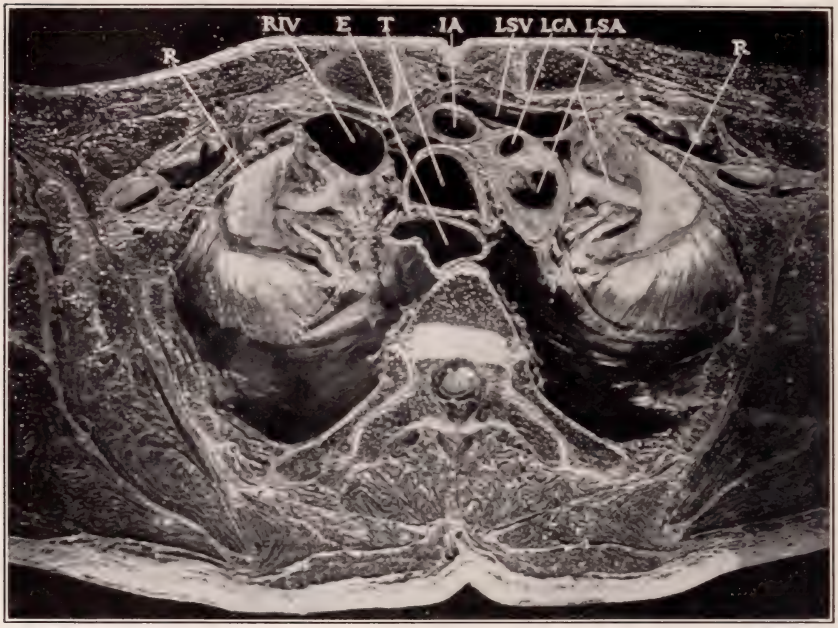

FIG. 6.—Horizontal section of the thorax just above the sternoclavicular articulation. The pleura has been dissected away. This photograph shows (*a*) the anterior position of the innominate artery, (*b*) the posterior position of the left subclavian artery, and (*c*) the anterior position of the right innominate vein. *R*, first rib; *RIV*, right innominate vein; *E*, esophagus; *T*, trachea; *IA*, innominate artery; *LSV*, left subclavian vein; *LCA*, left common carotid artery; *LSA*, left subclavian artery.

The same principle applies to the veins. The right apex is encroached upon and markedly grooved by the subclavian and innominate veins, these grooves being continuous with one another, and situated in front of the groove for the subclavian artery. On the left side the subclavian vein produces a sulcus in front of that of the artery, but being more mesial it does not encroach to so great an extent on the apex.

Second. The smaller size of the right apex as compared to the left. This is dependent on the facts enumerated in the previous

section. On the right side the vessels pass in front and occupy space which on the left side is occupied by lung tissue.

The shape and size of the pulmonary apices are modified by the vessels in the same way as would be two small hills over which roads were cut. In one case, as on the right side, the road is cut over the side of the hill and requires excavation. On the other side, corresponding to the left apex, the road is carried more nearly over the summit and comparatively no excavation is needed. The result, as far as the apices are concerned, is to cause the former to be smaller than the latter and to give the right apex a more conical, and the left a more dome-like shape.

Third. The apposition to the inner surface of the right apex of the trachea and to the left of solid tissue. This has been emphasized by one of us in a previous paper,[1] and is demonstrated clearly in Figs. 4 and 5.

CONCLUSIONS

1. Dextrality of the individual has no effect on either the vocal resonance and fremitus or on the percussion pitch and resonance at the apices.

2. The percussion note in the healthy individual is practically always less resonant and higher pitched at the right apex, except in the presence of unusually well-developed left pectoral muscles, and then only if very light percussion is employed.

3. The normal differences are due to the anterior position of the large vessels in relation to the right apex, as compared with the left, to the consequent encroachment upon and reduction in size of the right apex, and to the contact of the inner surface of the right apex with the resonating trachea, while the left is in contact with non-resonating solid tissue.

4. The differences are most marked anteriorly and mesially, because in this situation both trachea and vessels would exert their greatest influence, less marked posteriorly and mesially where the influence of only the trachea would be present, and least marked

[1] Loc cit.

laterally, since the outer portion of the apex is farthest removed from the trachea, and the vessels at this point are practically similar in their relations on the two sides.

We wish to express our indebtedness to Dr. George A. Piersol for anatomical material placed at our disposal, to Dr. R. Tait McKenzie for the privilege of examining a large number of left-handed students, and to Dr. P. G. Skillern, Jr., for assistance in preparing our anatomical specimens.

DISCUSSION

Dr. James Tyson: While I have been familiar for many years with this difference in the physical signs, particularly percussion and auscultation, at the right apex as compared with the left and with the explanations commonly offered, they never seemed to me entirely satisfactory. I confess, however, that I have never made any effort to arrive at a more satisfactory explanation. I fear I belong to that class mentioned by Dr. Norris as those satisfied to accept results without further investigation. I feel now that Drs. Norris and Fetterolf have reached an explanation which is, up to the present time, the most satisfactory.

To refer for a moment to one or two of the other explanations, as for instance that which ascribes the impaired resonance at the right apex to the pectoral muscle. The seat of this higher pitched and diminished resonance is above the spread of this muscle. The nearer you come to the clavicle the more striking these differences are. That explanation therefore cannot hold. It would seem evident to anyone giving the matter a little thought that there must be some substance interposed in that situation which raises the pitch to percussion and prolongs the expiration by facilitating conduction. The explanation which ascribes these changes to the distributions of the right bronchus is not satisfactory because there is nothing associated with it which can improve conduction. On the other hand such a distribution might reasonably be expected to give increased resonance to percussion and lower pitched breathing sounds.

Dr. Hobart A. Hare: Undoubtedly the writers of the paper are familiar with the research carried on by Dr. Charles Cary, of Buffalo, reported to the American Association of Physicians, about twenty years ago. In this work Dr. Cary made a study of the conditions of the right apex by running into the lung an alloy metal which had a low melting

point. It therefore would not damage the lung. My recollection is that Dr. Cary's conclusions, while not identical with those of the writers of the present paper, in making the trachea the chief factor involved, proved that the commonly accepted idea concerning the bifurcation of the right bronchus was not an adequate explanation. The quotation by Dr. Norris of the paper of Dr. Flint before the College brought to my mind the fact, familiar doubtless to many of the Fellows, that this was the elder Flint, and that he made the demonstration with loaves of bread, fresh and stale. From the fresh loaves a dull note was obtained; from the stale, a tympanitic note. When Dr. Flint took his seat, the equally great surgeon, the Senior Gross, jokingly asked him whether all New York bread was "tympanitic?"

Dr. James M. Anders: There are just two points I wish to mention. One is that, judging from the pictures shown here to-night, the difference in size of the two apices is so slight that it probably has very little to do with the difference in their percussion notes. The other point to which I would refer is that while the discovery made by Drs. Norris and Fetterolf with reference to the anatomical arrangement, especially of the blood-vessels on the right side compared with the left, is probably the true explanation, or at all events the major factor in explaining the difference in the note on percussion, still it seems to me that we should not lose sight of the facts that the right main bronchus is the larger, that it lies more superficially and somewhat higher up in the chest than the left and gives off numerous branches, all of which air-containing tubes occupy space on the right-hand side which on the left is occupied by air cells. This factor cannot be entirely eliminated, since it must add a tympanitic element, although as compared with the difference in position of the vessels of the two sides, shown by the results of the studies of Drs. Norris and Fetterolf, it is probably unimportant.

Dr. Robert N. Willson: I want to congratulate Drs. Fetterolf and Norris on their very beautiful photographs. I think there are very few, and only those who have done this work, who know how difficult it is to prepare sections and photographs of this kind.

I hesitate to offer still another explanation of this change of note. There is, however, one other which I think should be suggested as at least actively contributing to the conditions under discussion, and it has undoubtedly occurred to many. I think that anyone who tries to inflate his own chest will find that he can and does inflate his left chest much more fully, easily, and completely than his right. I think it is a well-known and recognized fact that the upper part of the right lung is, as a rule, less fully inflated than that of the left lung. It is much more often involved in diseased processes for the reason that much more often there is stagnant

and easily infected and infectious air present. It is also well recognized that very frequently the extreme right apex is involved with old scar tissue. The autopsy table shows in a great many fairly healthy lungs old healed or latent tuberculous foci. Both of these conditions of necessity must contribute toward the difference in resonance and pitch of the note on the right side, especially of that portion above the third rib.

I feel much as Dr. Anders does with regard to the failure of the photographs as shown tonight to demonstrate the markedly contracted air-filled lung space on the right side above the third rib as compared with that on the left. Indeed, these photographs appear to demonstrate exactly the opposite condition as the rule rather than the exception, the right apex appearing fuller and more dome-like than the left. Many lungs which I have removed with the heart show little difference at the extreme apex between the right and left sides. It would appear that the only difference in the percussion note that can be laid to the anatomical explanations offered to-night would be one based on the position of the large vein placed over the anterior surface of the pulmonary apex on the right side. That this cannot and does not constitute the only causal influence in the light of clinical and postmortem study seems a matter for almost certain common agreement.

Dr. Norris closes: I am afraid I have not made our position entirely clear. I should have stated at the outset that when I spoke of the right apex and the left apex I did not refer only to that portion which lay above the clavicles, but also that portion of the lung which extended down as far as the upper border of the third rib. I think that this statement alone will, if considered, tend to modify certain criticisms which have been made regarding our statements as to the relative size of the two apices.

The pictures of lungs shown were prepared from specimens used by Dr. Piersol in his *Anatomy*, and were made from frozen sections. In ordinary autopsies in which the lungs are more or less collapsed and misplaced the relations are disturbed, and as with all other viscera it is only by means of frozen sections that we can judge of relative positions. Regarding what Dr. Anders has said, we have failed to find, utterly and entirely, this difference in the anatomical distribution of the bronchi to which he has referred and to which allusion is made in many of the text-books.

THE COLLEGE OF PHYSICIANS AND THE PUBLIC HEALTH[1]

By JAMES M. ANDERS, M.D., LL.D.

The College of Physicians has been intimately associated with the developmental history of our city, and its founders clearly recognized the true relationship of medicine to the public health. Indeed, it may be confidently stated that the medical profession of Philadelphia has ever been found to the fore in the progress of civilization, and perhaps at no previous period has our profession as a whole been so deeply imbued with the importance of giving serious attention to sociologic and economic questions in so far as they are affected by hygiene, sanitary science, and preventive medicine as at the present day.

But though the birth of the College lies in the closing period of the eighteenth century, it has from its inception manifested an educational spirit based upon broad lines. Among its founders appear the names of some of the most famous physicians of colonial times—men who participated actively in the establishment of such conservative, leading, and enduring institutions as the University of Pennsylvania and the American Philosophical Society.

The College of Physicians of Philadelphia "was modelled upon the lines of the Royal College of Physicians of London, and consisted of a body of men of the highest professional standing, who met at stated intervals to read and discuss scientific papers and to consider questions of public health."[2] During the earlier

[1] Read February 7, 1912.

[2] The College of Physicians of Philadelphia, Founders' Week Memorial Volume, page 126. J. Norman Henry.

years of its history, most probably some of the measures proposed by it, intended to benefit the laity, were mere cloaks for professional ignorance. Nevertheless, a well-sustained effort was almost uninterruptedly put forth to safeguard the public health by timely suggestions, appeals to the Legislature, and by petitions and protests to the local authorities.

During the first year of its long life the College memorialized the Legislature, "Setting forth the pernicious effects of spirituous liquors upon the human body and praying that such a law may be passed as shall tend to diminish their consumption." Again, while yet in its swaddling clothes (1789), the College was requested by the Legislature to furnish it with information relating to the subject of the prevention of the spread of infective diseases.

In 1793 the College addressed to the Mayor a series of recommendations concerning the severe epidemic of yellow fever then prevailing in the city, while later on, or in November of the same year, Governor Mifflin requested it to furnish him with information in respect to this disease, especially as to its origin, cause, and the best methods of stamping it out. This request brought forth a reply, prepared by a special committee of the Fellows of the College, in due season. The College manifested a similar sacrificial spirit throughout the epidemics of 1797 and 1798, and it is a matter of authentic record that not only the Governor, but the Legislature and the local Board of Health repeatedly requested assistance and counsel in the discharge of their several official duties.

These are but few of the many instances in which the College, during its early history, availed itself of opportunities to promote the health and well-being of the general public. It was commonly appealed to at that time for aid and counsel by State and local authorities, and held a more commanding position relatively in regard to public health and civic questions than it does at the present day.

True it is that immediately subsequent to the year 1808 the College became inactive for a number of years, and thereafter, from time to time, passed through similar lethargic intervals,

but, on the whole, it has witnessed a remarkable growth in influence and standing as a scientific body. The College has also expanded its Mütter Museum to a commendable degree (the nucleus being the private collection of Dr. Mütter, which he donated), and has grown a library since it removed in 1863, into the then new building at Thirteenth and Locust Streets, of which the profession of any municipality might feel justly proud, and one of which but few, if any, can boast. Again, in regard to many public questions affecting the health and welfare of both the profession and laity, the College has made, on the whole, a fairly good showing.

Admitting these facts, may it not be reasonably claimed notwithstanding, that the College has failed especially in recent years to give as much consideration to questions of public health as has been required by the professional tendencies of the times, or, as its founders hoped would be given by their successors—by us, and as they actually gave, to them. The College has numerous "Sections," but not one on "Hygiene, Sanitation, and Preventive Medicine;" it has numerous standing committees, but none covering these important subjects.

It is not my purpose to criticise the affairs of this great institution, but simply to invite attention to an avenue of effort through which the College might still further increase its sphere of usefulness in conformity with the views and aims of its illustrious founders.

It might be argued that the many organized agencies, municipal and State, operating in the interests of the public health, meet every need in this direction at present; but I feel strongly that the time has come when the rank and file of the medical profession cannot afford to be lacking in knowledge of or interest in hygiene, sanitary science, and preventive medicine, and when toward the accomplishment of this end, medical associations must co-operate actively and systematically. Says Jacobi, pertinently, the up-to-date, present-day physician is a sanitarian.

Opportunities constantly present themselves to physicians for improving the hygienic affairs and conditions of the individual

or household, *i. e.*, in the direction of personal and domestic hygiene; but public measures of far-reaching sanitary import and essential to meet certain indications, which are beyond individual effort, must be considered, suggested, and executed by medical or other organizations, local health boards and the like.

The College as the result of a slight enlargement of its present activities could arrange for a systematic campaign of education among the laity, which is eager to be informed on health topics. The object should be to furnish just such practical information, bearing on disease and how to prevent it, as is essential to the public welfare. It seems to me that one of the best methods of carrying on a campaign of education for the benefit of the people at large would be by authoritative popular lectures to be delivered in this Hall. In this way it would be possible for the College to mould public opinion with, oftentimes, crystallization into suitable laws and sanitary regulations. The College should also play a conspicuous role in bringing about the introduction of improved training in hygiene and sanitation in the public schools, and the establishment of chairs of hygiene and sanitary science, in the public schools for the advanced pupils, in our academies, scientific colleges, and universities, as well as in our medical colleges. It is a serious question whether students of medicine receive adequate training in these subjects, although this is doubtless true of many of the better medical colleges of this country.

Again, by the creation of a standing committee on hygiene, sanitary science, and preventive medicine much good work could be accomplished without any serious interference with the usual scientific proceedings of the College. Such a committee would naturally keep in touch with and report on all questions of especial importance concerning the status of public health matters and would also recommend methods and means of dealing with the same to the College, which would be approved and executed if concurred in by this body. In this way a bureau of important research work pertaining to questions of hygiene, sanitation, and preventive medicine could be established and authoritative

opinions issued to the community at large, from time to time, bearing upon these subjects.[1]

Finally, it seems to me there should be no unwillingness to modify the present lines of activity of the college so as to render it more effective in this field of endeavor, more particularly since this would be in consonance with the aims and purposes of the founders of the College. The College has recently widened its boundaries, and included in its fellowship are men well fitted to conduct labors in the domains of hygiene, sanitary science, and preventive medicine, and I confidently believe that they could institute successfully a new section covering these subjects. Moreover, this would be in harmony with the trend of opinion in the profession at the present day.

Organizations, both medical and lay, are everywhere being formed for the purpose of precluding the possibility of the occurrence of disease, and already the character of the general practitioner's daily work has undergone a truly remarkable change as the result of the health movement now in progress.

It is from the line of effort herein outlined that physicians should derive their greatest satisfaction and enjoyment, and organized bodies, such as the College, compass the fulfilment of their noblest achievement—the preservation of the health of the community.

DISCUSSION

Dr. A. C. Abbott: As a literary product and comprehensive treatment of the subject I regard Dr. Anders' paper as finished, though brief. I regard his paper as most timely, and I believe the time ripe for the College to follow some such suggestion as has been made. I cannot but feel that the forward movement in preventive medicine should be taken not by laymen, but by medical men who naturally have a more comprehensive

[1] Since writing the above I have observed that a committee with similar objects has been formed by the New York Academy of Medicine. This committee proposes to undertake public health work on broad lines of activity.

and broader view of the subject; but the fact remains that in nearly all of our large and influential bodies organized for the welfare of the people the majority are laymen. In the Committee of One Hundred created for the purpose of forcing a Federal Health Department the minority are medical men. You will find on investigation that the medical profession is lax in its activities in many public health questions. From personal experience in public health work I am convinced that the authorities of this city would welcome the aid they might receive from an advisory body instituted by this College. I am very much gratified at hearing Dr. Anders' paper, and I hope the College will act favorably on his suggestions and thus perpetuate activities for which it has been in various degrees noted since its earliest history.

The President: The chair, after calling attention to the fact that in his annual address he had referred to Dr. Anders' paper, said that he was glad that the Fellows of the College had had an opportunity of hearing it. The chair then briefly reviewed the work of the College in sanitary matters, not only in its earlier career but in more recent times, and pointed out that after the development of the work of the State and County Societies in this respect, the College, as the honorary librarian had pointed out, had been less active in its relation to public affairs than in the earlier days of its existence, but, although this activity had been less conspicuous, the College had by no means forsaken its duties. The chair then referred to various public matters in which the College had taken an effective interest, some of which had come before the College on that very evening. He highly approved of the recommendation which had been made by Dr. Anders and also by Dr. Abbott that a committee on sanitation should be formed in the College, to which work of this character could be referred for consideration and action.

Dr. Richard A. Cleemann: I would say that upon all occasions of epidemics here the College has appointed a committee and we have had several very valuable reports. I do not entirely understand to what the Chair referred as taking the place of the College.

Dr. de Schweinitz, replying to Dr. Cleemann's inquiry: The chair quoted the paper of the honorary librarian when he stated that the College had been less conspicuous in its relation to public affairs after the establishment of the State Medical Society and the Philadelphia County Medical Society than it had been in previous years.

Dr. Cleemann, continuing: I think the College has been quite as prominent as any other society in Philadelphia in health matters.

Dr. Anders closes: The object of my paper was not to show that the College has not taken any interest, but that it should give more attention to the subjects of hygiene, sanitation, and preventive medicine in the

future than it has done in the recent past. I feel strongly that the voice of this College would receive very respectful attention from the general profession and the laity, as well as from sanitarians concerning these subjects on account of its influence, dignity, and recognized importance as a scientific body. It seems to me that the time has come when the College should reach out and become as potent for good in the domains of sanitary science and preventive medicine as it is and has been in those of scientific and practical medicine.

THE MEDICAL TREATMENT OF EPILEPSY[1]

By S. WEIR MITCHELL, M.D., LL.D., F.R.S.

In opening the debate on a subject so difficult to confine within set boundaries as that with which we deal this evening, I propose to myself the strictest limitations. My desire is to know whether any others have been more successful than I in the treatment of epilepsy, and whether it is not desirable to pursue investigations along lines other than those which have been followed.

Our subject this evening is the *treatment* of epilepsy, which of course involves more or less of its diagnosis and enables me to set aside at once all cases of distinct trauma, where surgical interference becomes desirable. There remain for consideration all those cases of epilepsy which are various in type and are known as idiopathic, a term conventionally employed, and which, it is to be hoped, may some day be set aside.

Treatment includes: First, the suppression of the *symptom-complex* by the use of drugs or other forms of general treatment; and secondly, the treatment during the brief period of the aura, which sometimes affords an opportunity to cut short the sequence of symptoms.

Let us deal with the first. We must, I am sure, feel a sense of therapeutic despair whenever a case of ordinary epilepsy appears for treatment at a clinic—nor does wealth make the situation much better. I must frankly admit that none of the remedial measures we apply for the general treatment of idiopathic epilepsy has any firm foundation upon reason or trustworthy experimentation on the lower animals. No man can tell me today why

[1] Read February 7, 1912.

bromides prevent epileptic explosions; nor is there any theory as to the causation of epilepsy which is not contradicted by some of the phenomena of this puzzling disorder. Most rare are reflex epilepsies; I have seen but two, once from a bean in the nose, and once from a crushed testicle. Both were cured by operation, and were reported by Hinsdale. It is conceivable that in nerve centres normal or abnormal substances may accumulate until they result in irritative symptoms and discharges of neural energy. But how then could this sequence be arrested by a mere sensory stimulation, like a ligature on an arm, or by abruptly dilating the cerebral vessels with amyl? The explosions would only be put off for the minute; the activating poison would remain.

The ingenuity of therapeutics has become bankrupt in the effort to find drug cures for epilepsy. It is needless to run over them, and I limit myself simply to the statement of certain conclusions. One of these is that the bromides are useless in certain cases of the minor epilepsy (petit mal), which respond favorably to some of the coal-tar products. This was not an uncommon experience in my clinic, and has been also observed in that of Dr. John K. Mitchell. Then there are examples of the lesser epilepsy which are made much worse by bromides; and also there are rare and inexplicable cases of the greater epilepsy in which bromides make the convulsions more violent and increase their number.

I may say that I have not found any great difference between the various bromides, such as has been asserted by some French observers, nor any special value in mixing them. For rapid action and for soothing purposes, I am of the opinion that the lithium bromide is the best. I state this with the doubt of self-criticism, because I myself introduced it into medicine. It is, however, the only deliquescent bromide, and contains the largest percentage of the agent bromine, which seems to be that which gives value to these salts.

It would be useless to talk to a set of men such as are here today in regard to the management of the doses of the bromides. Their deleterious effects are well known, and it is desirable, of

course, to give just that amount of bromide which being added grain by grain on the return of attacks will finally prevent the occurrence of any attack. During this process of gradual increase you run against the occasional difficulty that effective doses, varying with the individual, may produce loss of memory, inertness, low spirits, and in rare cases forms of positive melancholia, maniacal excitement, and even suicidal or homicidal mania. You may play all the tricks you will, using arsenic, hot baths, and what not, and still you will sometimes be driven to conclude that a man had better have epilepsy than be overdrugged with bromides. Of course, where the attacks come at long intervals this is the obvious course.

Although the effects of bromide on the skin are annoying, they may generally be avoided if the person will bear full doses of arsenic and use hot baths. There is, however, a small percentage of persons who are so susceptible to the bad effects of these drugs that a dose of a dram a day will within a week produce large rupial-like ulcerations, which so strongly resemble the rupia of syphilis that when I first saw and described them I was for a time completely deceived.

As you patiently add grain by grain to attain the restraining dose, so must you at the end of two years without attacks as slowly withdraw the drug grain by grain.

I am saying nothing about other drugs, but there is one thing I do desire to say. I saw, when young, cases of epilepsy cured by nitrate of silver, but the nitrate of silver had to be given in doses which would astound the therapeutist of today in order to bring about the result in question. I recall a case in which I did cure the epilepsy, being a rash young man, by large doses of the silver salt, having previously obtained a written promise that no action would be taken if I blackened the man. He was cured, and blackened.

There is one salt of silver which will not change under light, and that is the ioduret. Whether it will be efficient in epilepsies to bring about good results, or will cause such gastric troubles as to prevent its use, has not been sufficiently tested.

I have avoided the luxury of the relation of personal cases and beg you to consider that every statement I have made has strong and absolute foundation in observation and well-noted facts.

To turn now to the immediate prevention of individual attacks of epilepsy. I myself believe that epilepsy always originates in sensorial centres with a discharge from these centres, which is or is not recognized by consciousness. Such sensory discharges are referred to exterior organs, as when we have the sense of an aura rising up from the abdomen, or beginning as numbness or what not in the hand, or arising in the form of optical auræ or of olfactory or auditory impressions. In other words, I believe the aura always exists, but is unperceived in most cases, owing to the suddenness with which the motor discharge follows. I want to make this matter clear to you, because I do not think it has been fully enough expressed in this form elsewhere; or, if so, will bear repetition. I regret not to be able to give you all the evidence upon which it rests, but I think a single statement will make it clear to a set of experts such as are here present, and will make some of you wonder why it has not been more fully recognized. It is supported by the fact than an epileptic having distinct auræ may go on with lessening of the interval between the onset of the auræ and the convulsive attack and loss of consciousness until the time between becomes too brief for memorial registration or any useful intervention. I hope that I make myself clear and that you will perceive the importance of what seems to me to be a novel thought, open to attack, and perhaps to defeat, by larger observation; but so far as treatment is concerned, it was necessary to speak of it.

Before mentioning the use to be made of the auræ and what opportunity they offer in a small percentage of epileptic cases, I want to say a word as to a set of phenomena which have been I think improperly classed, especially by Oppenheim, as also being of the nature of auræ. Thus, there are many persons who are habitual epileptics, who, especially after the disease has existed for a considerable time, have within a day before the attack

certain definite sensations or conditions, which are occasionally of value in enabling them to anticipate and be ready for the attack. To illustrate: Certain people before an attack, a few hours before or a day before, develop either a condition of slight excitement or a state of sensation of being agreeably well, which the French call *bienaise*, or well-being to put it in good English. You are familiar with the fact that this feeling occasionally precedes certain headaches. A more distinct symptom is the curious personal odor which in some cases precedes an attack, and may take the unpleasant form of horribly bad breath, which is unfortunately also one of the annoyances due to the use of bromides. This which I speak of now is described as being different, usually as having an acid odor, but the chemistry of odorous emanations has yet to be studied. There are various other forms of the general or local states which precede the aura and the fit, but I do not propose to dwell upon them here. I speak of these prodromes only because the therapeutic opportunities they present are often overlooked.

If now we are to help a person in the few moments which precede the epilepsy, two or three things are necessary, and they unfortunately exist only in a moderate percentage of cases. These are: An aura long enough to permit of something being done, capacity on the part of the sufferer during that time to use this interval, and the dicovery of what is the best form of interference. These interferences to be valuable are all, with perhaps one exception, such as affect the sensory stage of the connected series of symptoms called epilepsy. Some of them are as old as knowledge of the disease—that is, such as putting salt on the tongue, a ligature around the arm, and the like. I shall mention only those which are novel in their mode of employment or in kind.

If you have to deal with an epilepsy of Jacksonian type or originating in a sensation confined to one limb, a sudden ligation of that limb will very frequently arrest the attack. How to bring this about when the aura is brief is the question. I succeeded very well in one remarkable case, by having a spring arrangement worn on the left arm and set free to act by pulling a cord which

was fastened at the wrist. A sharp pull on this cord released the spring and pretty violently clamped the arm. This usually resulted in checking the attack.

The two other points to which I shall now allude are: First, that a great change in the mode of life sometimes results in curing epilepsy. Thus I have known two cases in which service in a cavalry regiment during the Civil War did put an end to epilepsy. I have made all sorts of efforts in the way of diets, starvation, the milk cure, the meat cure, absence of salt, rest, and what not, without much good. Generally the burden of unhappiness is needlessly increased by forbidding tobacco and coffee. I have had the frequent experience known to others that a change of remedy or locality, such as putting a patient in a hospital to study the every-day phenomena of attack, were followed by long periods of arrest of the disease.

And what of the use of nitrite of amyl in the treatment of the attacks? This, I am perfectly sure, will arrest attacks of epilepsy if the aura gives time and opportunity. Neither need there be any fear in the use of this drug, although the sensation it causes in the head is alarming. I have seen it pretty recklessly employed by a rough hospital nurse, with the effect of stopping successive attacks in the status epilepticus. When I first used this drug in 1871 (and I was the first to employ it) I personally used it in an attack I witnessed, and was completely satisfied of its extraordinary power. My paper appeared in 1872, but it has since been rediscovered by Crichton Brown and one or two other people, about which I have not further concerned myself.

The condition known as minor epilepsy (petit mal), which sometimes lasts for minutes, is not as easy to end by the use of amyl nitrite as is the aura of the greater epilepsy.

I have not dealt here with the reason why the nitrite of amyl cuts short the series of phenomena which accompany the onset of attacks. The great pallor of face which ushers in the fit suggests how it acts, and this alone is sure, that if you can thus or in any way bring on flushing and dilatation of the arterioles you may so interrupt these sequences as to prevent the storm of convul-

sive movements which would have followed. Also, it is true that if we can control often enough the occurrence of convulsions we may lessen the probability of their continuance or recurrence. Apparently we may break up what one crudely calls the convulsive habit.

Finally, there is one other matter which I consider important. It is not my discovery. I do not now remember who told me of it, but it was a physician I met somewhere on a train in New York State. He told me that having been subject to a grave form of epilepsy, preceded by very horrible olfactory subjective odors lasting fifteen or twenty minutes, he discovered that by taking a long inhalation, holding his nose, and then forcibly contracting the chest he could check the progress of the aura and prevent altogether the convulsions which without this measure inevitably followed. He assured me that he had thus succeeded finally, without bromides, in altogether stopping the epilepsy, and he no longer employed or had need to employ the method in question. This is almost my only useful contribution this evening, and I think it worth while to state it, because I have seen a number of patients able to relieve themselves by this means. If you will employ it on yourselves you will see that it flushes the face, and therefore in all probability flushes the brain just as amyl does, and thus interrupts the sequence of events which it seems must develop in turn before the convulsive symptoms occur. I had a letter last week from a lady who by this means, having a long period (several minutes) of remarkable aura in the forms of rings of light before her eyes, has been able to check all her attacks during a year with one exception. I ought to say that she finds she is thus able to get along with one-third of the quantity of bromide which before was necessary to prevent recurrences and which left her a wretchedly melancholy person with increasing difficulty of locomotion. I therefore suggest in these cases a moderate amount of bromide, with some effort to use the method in question. It is one which requires a little training.

I think that I have here said everything which it is worth while for me to say, but desire to add that with the exception

of these slight hints as to the auræ, I want most to direct attention to the fact that probably *all* attacks have auræ and *all* attacks have a sensory origin.

To conclude, I ask myself and you, What is the effect on the brain cells of excessive use of bromides? I ask also is it not possible that they act locally on the vessels of the sensorimotor areas so as to prevent accumulation of a convulsant substance, and so give time for its slow elimination? My question is purely hypothetical, but admits of some defence, and in fact epilepsy offers many unanswered questions.

THE MEDICAL TREATMENT OF EPILEPSY[1]

By WILLIAM T. SHANAHAN, M.D.

In presenting this paper on the medical treatment of epilepsy, I can but endeavor to review concisely what has been done in the past in the therapeutics of this condition and summarize what, to me, seems to be the best general method of treatment now known. Naturally, many drugs and methods have had their day and then passed into deserved oblivion. Others, although long known to the profession, have not always received the proper recognition which their importance should have gained for them. In a paper of this length many of these things must necessarily be referred to in the briefest manner. The surgical treatment of the disorder will, I believe, be discussed by other speakers of the evening.

No theory ever put forward to explain the origin of epilepsy has been proved in such a way that an exact scientific treatment could be pursued. Thus empiricism continues naturally to play a major part in the therapeutics of the condition.

If we accept the hypothesis that epilepsy is due to the absence of a protoplasmic factor that determines complete nervous development, we are in a position where, if we expect to arrest or cure, we must either supply this missing factor or so modify the relation between the factors present as to have the functions of the organism as nearly normal as possible. The former is impossible, at least with our present knowledge; the latter seems attainable to a certain degree.

[1] Read by invitation, February 7, 1912.

To assign bacterial infection as the exciting cause of the symptoms of epilepsy, as did Bra, seems not worth giving even consideration. Much more credible evidence exists that an abnormal or perverted metabolic activity is present in the epileptic, in consequence of which certain chemical substances, either from the gastro-intestinal tract or from one or more of such glands as the pituitary, adrenal, parathyroid, thyroid, pancreas, or similar structure, become active toxic agents, as a result of whose influence on the central nervous system the phenomena of epilepsy appear.

Research has thus far failed to firmly establish a logical chain of facts explaining beyond question this supposed perversion of function in its relation to epilepsy. If time permits the unveiling of accurate knowledge covering these matters, we can look forward to our having at some future period a specific treatment for what we now designate as idiopathic epilepsy, or more properly speaking, epilepsies.

Although the underlying factors, such as developmental defects or changes consequent upon early meningitis or encephalitis, whether due to injury at birth or subsequently may be permanent, the actual exciting cause of the seizures may be eliminated by either preventing its formation or by rendering negative its pernicious influence.

In comparing the results obtained from treatment in institutions and that outside, we must remember that the majority of the former cases are ordinarily of several years' duration before admission, whereas many of the latter are observed shortly after the onset of the condition, and, other things being equal, are of a more favorable type for treatment.

Camp[1] states that a cure of epilepsy may be reasonably expected "when the primary cause is known and removable." How true this is. We must confess, however, that in the great majority of cases it is impossible to accurately ascertain this primary cause.

It has long been remarked that in the treatment of epilepsy all remedies do good for a time, this being due in part to the

psychic influence on the patient. It is a common experience to observe, after a period of freedom from the disease that varies from weeks to months, and while treatment is being faithfully employed, to have the symptoms recur.

Epilepsy is such a broadly inclusive term that it involves conditions which differ markedly so far as their response to treatment is concerned. Where the symptoms are due to focal organic disease, overindulgence in alcohol, arteriosclerosis, etc., it is logical to assume that early treatment of a proper nature may reasonably be expected to bring forth better results than where the patient has a generally defective make-up, a paralysis of long standing, or some other chronic condition.

We all know that frequently the symptoms of epilepsy have continued unrecognized for many years, thus permitting the convulsive habit to be firmly established. Idiopathic epilepsy, appearing as it does in 85 per cent. of all cases, before the twentieth year, shows itself as a disorder of the developmental period. This is another reason why permanent degenerative changes in the nervous system are the rule and not the exception.

In outlining a course of treatment for an epileptic, the physician must impress upon him and his family that inasmuch as epilepsy is a chronic disorder, the special treatment must continue over a long period, and that in fact the patient must, during the balance of his days, live according to certain closely drawn rules relating to hygiene and diet. Unless earnest coöperation is secured for the carrying out of these details, one cannot expect to obtain the desired results.

The physician himself must not relax his vigilance in the following up of his epileptic patients. Perseverance is indeed a virtue in the treatment of this condition.

The readjusting of the patient's mode of life is oftentimes a difficult process, and, of course, in many cases cannot be as complete as one would wish. The essential changes indicated in the individual case must, however, be insisted upon or it is useless to attempt to treat the patient.

When the occupation, means of recreation, the bathing, diet,

and hours of sleep have been properly regulated, a long stride has been made toward assisting the individual in acquiring a more stable functioning of his central nervous system. The initial effort required to bring about coöperation must be well tinged with pyschic influence, without which our means of treatment of nervous disorders would be small indeed.

The psychical influence in the treatment of epilepsy is of considerable importance, as a belief aroused on the part of the patient that he is to be helped will pave the way for a serious effort being made to carry out whatever treatment may be prescribed.

Hope and confidence must be aroused in your patients before you can expect to secure beneficial results from your treatment.

Prophylaxis in general must consist in continued efforts being made toward arousing a public sentiment which will demand the segregation at least of the epileptic who is mentally defective. We can never hope to have isolated the small percentage of epileptics whose mentality has not deteriorated. Before, however, anything can be accomplished in this general direction, physicians as a whole must be made to realize the results of the mating of defectives. The average practitioner does not appear to grasp the seriousness of this problem; such being true, how can we expect the general public to understand these matters as they should?

Relative to prophylaxis as applied to the individual, I cannot reiterate too much the plea that all convulsions in infancy and childhood be recognized as very serious occurrences, so much so that such a child requires careful supervision ever after. Parents and those who have to do with the raising of children must be brought to appreciate the great importance of giving proper attention to the diet and hygiene of a child in whom even one convulsion has occurred. Such care would, in many instances, result in these children developing into strong, healthy adults with no evidence of an unstable nervous system. Lack of such attention too often means recurrence of convulsions, and in consequence a progressive deterioration of the unfortunate individual.

With the exception of the sedative effect obtained by the dis-

covery of the effect of the bromides, we may say the general principles of treatment of epilepsy were recognized thousands of years ago. The importance of exercise, bathing, and the careful regulation of the diet was recognized by the earliest writers on the subject—in fact, so carefully were these methods laid down that we cannot at this day materially improve upon them.

We must confess that the medical treatment of epilepsy is essentially ameliorative and, comparatively speaking, of minor importance when one considers the value of strict dietetic and hygienic measures.

It is a matter of common observation that the natural tendency of epilepsy is to progress, rarely to cease spontaneously, after it is well established.

It has been well said that the subject of epilepsy is one that embraces, perhaps more than any other limited department of medicine, the whole range of therapeutics. Sieveking,[2] a writer of sixty years ago, stated: "In epilepsy the results have been, if not barren, yet unsatisfactory; and still the disorder constantly attracts new inquirers, each anxious that he may succeed in lifting the veil that shrouds the mystery. We are justified in expecting more decided benefit from a full development of our hygienic resources in combating epilepsy than by reiterated experiments with drugs. Epilepsy is a disease of the whole man and not of any one organ or system of organs alone. In many instances there is no doubt that the result of treatment is merely a temporary arrest or postponement of the affection, which is erroneously regarded as a cure."

Sieveking also tells us that "there is not a substance in the materia medica, there is scarcely a substance in the world, capable of passing through the gullet of man, that has not at one time or other enjoyed the reputation of being antiepileptic."

I must agree with Dr. A. J. Rosanoff,[3] who, after making dietary tests, concluded that the epileptic should receive the largest amount of carbohydrates and fats that he can properly assimilate and the smallest amount of proteids compatible with the preservation of the nitrogenous equilibrium—that is to say, the amount

of nitrogen ingested must not be allowed to fall below the amount excreted.

I scarcely need to refer further to the details of arranging a diet for the individual case. What one epileptic may use with impunity may be very harmful to another. The purin-free diet advocated by Turner and others has some value perhaps, but I cannot see that the results following its use differ from the ordinary restricted diet. It is easier to restrict the diet and to regulate the action of the bowels than to treat by intestinal antiseptics, although the latter may have some place.

The history of the use of the bromides from Sir Charles Lacock's advocacy of them in 1857 for use in epilepsy up to the present is so well known that I will not review it at this time.

The bromides' only use is to diminish the frequency and severity of seizures. The removal of the instability of the brain, if such is possible, must be accomplished by other methods. I would repeat that a careful regulation of the method of living is more essential in the treatment of epilepsy than is medicine. The bromides in epilepsy might be compared to a muffler or governor to control temporarily.

How frequently has the following observation of Peterson[4] been overlooked: "In order to give scientific value to any new therapeutic measure in epilepsy, it is necessary to show that the patient has not been using bromides for a long time before the experiment in the new treatment has been undertaken."

Sterile solutions of the bromides have been used hypodermically in status epilepticus, but to me this has always appeared to be the heroic use of a measure whose effect does not warrant such a procedure, as the control of the seizures, when such is possible, can be gained by other methods.

It has been aptly said that the use of bromides results in a chemical restraint of nerve cells. It has also been asserted that if the bromide is continued over a long period, chromatolysis of cortical cells and even dissolution may occur.

Delirium as a result of the overuse of the bromides is a symptom which, when it occurs, may not be ascribed to its proper source.

I will but mention hypochlorization, as Toulouse's method of administering the bromides is too well established to need my commendation.

For those having seizures shortly after arising, it would be well to give a sedative immediately upon awakening, having the patient take a light breakfast in bed and remain lying down for from one-half to one hour. After this period the readjustment of the cerebral conditions from their state during sleep to that during waking hours has occurred, and the patient may arise.

Where seizures occur during the first few hours of the sleep state, the sedative should be given two or three hours before retiring and following a light supper.

In many cases seizures should be understood as occurring not necessarily at night but *during sleep*, whether day or night.

Bromine may be administered as one of its salts or a combination of the same, being well diluted. The numerous vehicles prescribed disguise the taste of the salt in varying degrees. More elegant preparations of bromine are bromipin, bromoglidine, brometone, sabromin, brovalol, etc. In my experience sodium bromide is the most satisfactory salt to prescribe.

Vance[5] divided cases into two groups as to ophthalmoscopic findings: (1) One with vascular fulness, (2) the other with anemia of the retinal vessels; each finding showing, according to him, the state of circulation in the brain—*i. e.*, directly opposite conditions may give rise to the same symptoms. He claimed that bromides proved very efficacious where retinal congestion appeared and that bromides aggravated cases showing retinal anemia. In anemic retinal cases following several paroxysms, the retinal veins became dilated and tortuous; the arteries, however, remained unusually small.

The treatment of the epileptiform seizures, due apparently to arteriosclerotic changes in the cerebral vessels, consists in proper rearrangement of the habits of the individual, excluding so far as possible everything which would tend to raise the blood-pressure in these vessels. Iodides have a place in the treatment

of this type, but of greatest importance is the regulation of exercise, diet, and hours of rest.

In symptomatic epilepsy appearing in adult life, the first convulsion serves, oftentimes, as a warning and leads to careful treatment. Are such convulsions indicative of a latent convulsive tendency? The threshold value of stimuli necessary to induce convulsive phenomena must be lower in a part at least of these individuals. A great number of persons who never manifest such symptoms have as marked an arteriosclerosis in comparatively early life, and are also alcoholic or syphilitic, or both.

Epileptiform symptoms appearing during the course of some thyroid disturbance resulting in insufficiency of secretion of that gland might be expected to be relieved when thyroid extract was administered in proper dosage. To make a broad claim that thyroid extract should be given to all epileptics is absurd.

When epilepsy appears after twenty years of age, syphilis must always be considered in searching for the exciting cause. There is no pathognomonic sign peculiar to syphilitic epilepsy except possibly the Wassermann reaction; and even this, strictly speaking, has been obtained in leprosy, etc. Partial convulsions are presumably evidence of the seat of the lesion, being the expression of definite localization and thus prove symptomatic. In some cases, however, it would seem that the centres in which the convulsion seems to originate are affected from distant parts. These symptoms, as just stated, occur after the usual age of onset of idiopathic epilepsy and may result from syphilitic dyscrasia.

When a positive Wassermann reaction has been obtained in such patients, salvarsan should be at once administered, preferably by the intravenous route. Mercurial treatment to supplement this may be found necessary. After a varying period another Wassermann test should be made, and if positive, another dose of salvarsan given.

A relative constipation, or a day late to stool, as it has been expressed, is common to the majority of epileptics. The excre-

tion of waste products is incomplete, in consequence of which, in the epileptic, we have a state which, added to the hypersensitive nervous system, results in the symptoms known to us as epileptic. Drastic catharsis has been decried, but from our experience at Craig Colony it is most valuable.

The moral treatment to inculcate self-control, personal discipline, and strict rules of conduct is of the utmost importance in the therapeutics of nervous disorders, consequently holding true of the epilepsies. Worry and stress act in producing an increase in the number of seizures. The psychical condition so influences sleep, digestion, elimination, etc., that the patient is brought below par, and this manifests itself in these persons by the recurring seizures. It is a well-known fact that psychical disturbances—*e. g.*, worry over examinations, business matters, etc.—may create in supposedly normal individuals an upheaval in the functions of the alimentary tract, circulatory system, etc.

As to the question of the marriage of an epileptic there can to my mind be but one answer—and that an emphatic "No." The type of offspring of such marriages, the desertions which result, the extra burden on the family of the afflicted one, who as time goes on becomes less able to take a place in society, are everyday occurrences and make one ask, How long will these matings continue to be permitted?

When we are consulted by epileptics who hold positions of responsibility where the lives of others are under their control—*e. g.*, railroad employees—we should in justice to society recommend a change of occupation.

Delasiauve, in 1849, recommended the establishment of institutions for epileptics where a hygienic as well as a medical plan of treatment might be pursued. He calls attention to the fact that every infraction of temperance will induce a relapse. A peaceable and quiet life is best suited to the epileptic, exercise being very salutary. Galen and Hippocrates laid stress on bodily exercise. The history of the development of the public care of the epileptic is so well known that I will not revert to it at this time.

Silvestri,[6] comparing the results obtained from animal experimentation with clinical experiences, concludes that a lack of calcium is a prominent factor in the production of convulsions, and that supplying large amounts of calcium may render inactive the substance causing the convulsion.

Some observers report excellent results from the use of calcium lactate; others obtained no beneficial effects. At Craig Colony our experience was the latter one. It has been claimed that the efficacy of milk diet has been due to the increased amount of calcium thus brought into the system.

For the aborting of seizures great results have been claimed to be obtained by inhalations of nitrite of amyl to so modify the cerebral circulation by producing a dilatation of the vessels as to abort seizures. I have seen no beneficial results from other alleged methods of aborting seizures. It is my opinion that in such instances the seizures were of an incomplete type, and in consequence the apparent effect of the abortive measure was obtained.

The serum treatment advocated by Ceni, Mazzei, etc., has not proved to have the value claimed for it.

Our experience at the Craig Colony with female epileptics leads us to conclude that the alleged relation between the menstrual function and seizures is largely overdrawn. In several hundred cases it was found that there was no apparent connection between the two conditions. In a few cases mild seizures seemed somewhat more frequent at the menstrual periods than during other parts of the month, and in a few the seizures occurred just before or after menstruation.

The fitting of glasses, the correction of dental abnormalities, and other similar corrective measures are of value in that they tend to place the individual in a more normal state of health. It goes without saying that an ophthalmoscopic examination should be made in all cases. I have yet to be convinced that genuine epilepsy can be completely arrested by the correction of refractive errors or the removal of an unerupted tooth.

The importance of hydrotherapy in epilepsy is not always

appreciated. In a general way it is useful for its tonic effect. In a particular sense it is of value to bring about a greater activity of the skin and thus indirectly relieving the kidneys from part of their load of elimination.

Enemata given frequently and using large amounts of water are exceedingly useful in cleaning out the torpid lower intestine. The quantities of fecal matter which accumulate in the colon of many epileptics seems remarkable. In serial attacks and status epilepticus the colonic flushing is of the utmost importance. Its earlier use would in many cases have prevented the status.

Owing to the tendency of many epileptics to turn on their face during nocturnal seizures, they should use a small hair pillow, or better, have them accustom themselves to do without a pillow.

The accidents and injuries to which epileptics are exposed as a consequence of their disorder are multitudinous. Some of them will occur despite every precaution.

The school work of the epileptic child should be largely manual in nature. An important feature of all school work is of course the acquirement of method in doing things, or to express it in another way, the training along disciplinary lines. The teacher of the epileptic child requires a larger stock of tact and perseverance than perhaps with any other special class. I have seen many cases in which regular school work seemed to hold in abeyance, perhaps preventing, the mental deterioration so characteristic of epilepsy.

I wish to emphasize the fact that the suitable occupation for the epileptic is of prime importance. In this connection it is of interest to refer to the theory presented by Spratling that products from muscular action are capable of uniting with the unknown epileptic toxin and of rendering it inert. This might explain beneficial results obtained from properly regulated out-of-door exercise.

In the treatment of the special conditions such as status epilepticus, serial seizures, mental disturbances, etc., eliminative, sedative, and supportive measures with careful attention to nursing, are indicated.

To summarize: The principles of treatment of the epileptic are good, nourishing diet; sufficient exercise out-of-doors; a proper occupation, well balanced by a suitable amount of recreation; sleeping in a well-ventilated room, or, better still, out-of-doors; a regular amount of sleep; retiring early; avoidance of undue excitement or cause for worry; a minimum of medication; regular and frequent bathing; careful attention given to the organs of elimination. The confirmed epileptic should, without exception, live in the special institution where he is with his kind, and in consequence can be permitted privileges not available in the outside world. The seizures and mental status must both be considered in making the prognosis. Seizures may be controlled, but a mind which has deteriorated cannot be restored.

There is no more justification for any physician to definitely promise any epileptic he can be completely cured, to never have a return of seizures, than there would be in telling a patient who had recovered from pneumonia that he would never have another attack of that disease.

Pastor von Bodelschwingh, of Bielefeld, expresses it as his opinion that absolute recovery of the epileptic is rare, and that to consider an epileptic as recovered there must be a perfect restoration of the psychic functions as well as a cessation of seizures.

We have admitted to the Craig Colony for Epileptics during the sixteen years it has been established some 3460 patients. Some have remained for a very short time, others for the entire period; the average number have been with us for from six months to two years. There have been discharged during this same period as recovered 51 cases, and as improved 553, from a total of 2125 discharges. The percentage of recoveries, less than 2 per cent. of the total number under treatment, is observed to be low, but to me no lower than what must be expected with our present knowledge of the epilepsies and the great number with the incubus of hereditary defect of the central nervous system.

I may appear, perhaps, too pessimistic, but so many writers present such glowing reports of cures of epileptics that we who are familiar with the institutional cases cannot but be filled with

wonder at the results, and conclude that the class of cases described are of an entirely different nature from those seen in the special institutions. Spontaneous arrests occur infrequently, and are usually in cases which have had no special treatment other than the regular institutional life. In some alleged cases of cure one is justified in questioning the accuracy of diagnosis.

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THE MEDICAL TREATMENT OF EPILEPSY[1]

By F. X. DERCUM, M.D.

The term epilepsy embraces a number of symptom groups which, while closely allied, differ radically from one another as to their pathogenesis. Among them we have the (1) so-called idiopathic or essential epilepsy; (2) epilepsy due to the ingestion of poisons, such as alcohol and lead; (3) epilepsy the direct or indirect result of infection; (4) epilepsy due to gross organic disease of the brain, such as brain tumor; and (5) epilepsy the result of trauma. Obviously the consideration of the subject in its entirety is unsuited to an evening's discussion, and I will limit that which I have myself to say to a discussion of true epilepsy, *i. e.*, the idiopathic or essential form, the other forms being in reality merely symptomatic disorders.

When we study patients suffering from essential epilepsy, we are at once impressed by the fact that the larger number, if indeed not all, present the earmarks of retarded or aberrant development. It is hardly necessary to call attention to the peculiarities presented by the structures readily accessible to observation, such as the skull, the palate, the teeth, the ears, the limbs, the digits; nor is it necessary to point out that if anomalies, arrests, and deviations are present in surface structures, it is legitimate to infer that they are present in deeper structures as well. I presented many years ago before the Philadelphia Neurological Society a series of twelve brains of confirmed epileptics, all of which presented more or less striking anomalies of convolutions and fissures. It is very probable that other morphological

[1] Read February 7, 1912.

anomalies obtain elsewhere in the organism, and it does no violence to the facts to regard epilepsy in its essential forms as distinctly a morphological disease, as one occurring in defective organisms, organisms in which evolution has been incomplete or aberrant, and in which we have reason to believe that metabolism also is not normal and that the individual suffers from the formation and retention of toxic substances.

Time will not permit of a discussion of the facts bearing upon the theories of the toxicity of epilepsy. Studies in metabolism, as Sarbó[1] points out, while they promise something in the future, have failed to reveal any product of metabolism as the constant accompaniment or cause of epilepsy. However, as is well known, not only the seizures but also the symptoms of the intervallary period strongly suggest a toxic cause. Among the symptoms present during the intervals suggesting toxicity are mental depression and morbidity, emotional irritability, heaviness, slowness of thought, headache, sensations of weakness, and less commonly tremor and muscular twitchings. That, however, convulsive seizures may occur without toxic cause is proved by the convulsions artificially induced by A. J. Parker and myself in our own persons and in those of some of our colleagues. Our experiments were described in a paper read before the Philadelphia Neurological Society in 1884, and were also subsequently demonstrated to a small group of Fellows of the College. The method consisted in bringing about rapid nervous and physical fatigue by special strains, and while toxicity could not have played a role in the convulsions so induced, I notwithstanding believe that in the true epileptic convulsion the facts are in favor of toxicity. At any rate, in the treatment of epilepsy the factor of possible if not probable toxicity should receive serious consideration. Again, if in addition it be true that the epileptic is morphologically defective, this fact must for obvious reasons be equally borne in mind.

The treatment divides itself into a general or physiological treatment, and a treatment based upon drugs.

The physiological treatment resolves itself into the following

subdivisions: (1) Treatment directed toward the elimination of waste and toxic substances; (2) exercise; (3) diet; (4) the maintenance of the general physical and mental health as near the physiological level as possible.

Under the first head we should bear in mind that it is important to keep open all the avenues of elimination. The bowels should be opened once or twice daily; the kidneys should be kept active by the free ingestion of water, and the skin should be kept active by tepid sponge bathing or by brief tepid immersion baths followed by a gentle rubbing. The idea of the bath should be not to produce a reaction or a possible tonic effect, but simply to encourage the activity of the skin as an emunctory organ. Baths which produce profound reactions are contraindicated.

Further, it is important, I am convinced, for the patient to spend as much time as possible in the open air in order that proper oxidation of the tissues shall not be impeded. Equally necessary is it for the epileptic to sleep in well-ventilated rooms. Out-of-door sleeping I do not believe to be necessary, though of itself it is not to be decried. Exercise I consider of the utmost importance, but this exercise should be gentle; it should never be violent, and should never be of such a character as to surcharge the tissues and fluids of the body with waste products. Excessive exercise does harm. Overfatigue, as is well known, is a direct exciting cause of epileptic seizures. The indication for exercise in epilepsy is, (1) that the exercise shall be gentle, and (2) that it shall be taken in the open air; by it the intake of oxygen is increased, the oxidation of the tissues is promoted, and indirectly in this way elimination. Marked fatigue must always be avoided.

The question of the diet of epileptics is a very difficult one. The diet must be adapted to each individual case. Certain general principles, however, can be considered as established. First, it has been shown that in many cases the seizures are less frequent when the red meats, and perhaps the white meats also, are diminished, and in some cases when they are excluded. For instance, Alt[2] noted in studying his cases of epilepsy that the number of seizures became less pronounced when he restricted his

patients to a vegetable diet, and that they diminished most when they were upon a strict milk diet. In children he noted that the attacks became least frequent when the latter were given sterilized milk. Blumhardt[3] noted in twenty-one cases of epilepsy in children that a vegetable diet was followed by marked improvement. However, J. and R. Voisin,[4] basing their conclusion upon clinical experience, maintain that a meat or nitrogenous diet, no matter what the form of the latter, is not capable of increasing the number of attacks, provided the amount is moderate. Rosanoff[5] also concludes that a mixed diet does not exert any more influence upon the attacks than does a purely vegetable diet. He maintains that an exclusive vegetable diet in epileptics is not indicated. He noted, however, that either an excess or deficiency of the proteids had an unfavorable effect upon the attacks. He noticed the most unfavorable effects when there was an excess of proteids and a deficiency of the carbohydrates in the diet. The practical results of his investigations are that we are to give the epileptics as much carbohydrate and fat as they are able to assimilate, and as much proteid as is necessary to the maintenance of the nitrogenous equilibrium, but no more.

Lallement and Rodiet[6] studied in their epileptics the influence of diet on the amount of skatol and indican in the urine, and they compared their results with the results obtained after an ordinary diet. They found that with a vegetable diet there was a marked reduction in the amount of uric acid and chlorides. The indican disappeared almost completely. Skatol was, however, present in the same amount as in the mixed diet. On the resumption of meat, the amount of indican and uric acid again increased. Rodiet[7] noted a favorable effect of the meat-free diet on the mental condition of epileptics; they were less irritable and excitable than on a meat diet. Shanahan[8] insists upon a careful examination of the digestive tract and a study of the metabolism, examination of the bowel movements, and an analysis of a twenty-four hour specimen of urine. Baugh[9] claims that a diet poor in the purin bodies is not best for all epileptics, but is appropriate to only a part of them. Certain it is, however, that a rich purin diet exer-

cises an unfavorable effect upon the metabolism and the health of the patient. Rodiet, Lallement, and Roux,[19] who instituted in eleven epileptic women a strict vegetable diet, point out that it is incorrect to attribute the number of the attacks solely to gastro-intestinal autointoxication. They point out that the diet must be carefully adapted to the condition of the digestive tract.

In my experience it is wisest in epileptics not to adopt too strict and too rigid a diet. The diet should be a mixed one. It should contain vegetables and milk in liberal amount. It should contain the white meats. The red meats should be given sparingly. I have, however, occasionally found that if in epileptics who have been accustomed to a meat diet the meat be withdrawn absolutely, the number of the attacks is increased. In other words, some patients will improve with the diminution and others with the absolute withdrawal of the meat. With regard to the carbohydrates, I am myself inclined to adopt a middle course. I do not believe it wise to surcharge the intestinal tract of the patient with starchy foods. My experience is not in accord with that of Rosanoff, that we should give an epileptic as much starch and fat as possible, but that the quantity of starchy food should be moderate in amount. Inasmuch as the starches are the equivalent of sugar and are therefore muscle foods, and inasmuch as the epileptic patient is not to exercise strenuously, the amount of starches indicated is not large; further, starches in large amount favor digestive disturbances. As regards fat, a normal amount of fatty food may be given. As a rule the patient's own inclination can be trusted in this respect.

It goes without saying that in every case digestive difficulties should, as far as possible, be corrected, and especially should free evacuation of the bowels be insisted upon. I am not, however, an adherent of the theory of intestinal intoxication as an explanation of the epileptic seizure. In my own belief the cause of the autointoxication of epilepsy lies far deeper; it is possibly to be sought for in an as yet unexplained disorder of metabolism, possibly a disturbance of some gland of internal secretion.

There can be no doubt that by the carrying out of a rigid

hygiene—*i. e.*, by the keeping open of the avenues of elimination, by favoring the oxidation of the tissues, and by the avoidance of an excess of foods which hamper or encumber oxidation and metabolism in general—the number of the seizures is greatly influenced, and this irrespective of the administration of the bromides.

Time will hardly permit of an extended discussion of the occupation of epileptics. That the occupation should be simple in character and not involve nervous overstrain, that it should be carried out in the open air, that it is ideally presented by epileptics who spend their time upon a farm or garden, or in an epileptic colony, there can be no question. Every one of us has noted that nervous overfatigue, that sudden psychic or emotional excitement, may lead to an epileptic attack, and it goes without saying that the life of the epileptic should be made as placid as possible.

There can be no doubt that now and then simple physiological methods of treatment result in a lessening or even a prolonged remission of symptoms. I have had several such experiences. We must bear in mind also that every now and then, though rarely, epileptic attacks cease spontaneously.

In regard to the giving of medicines, I wish to state most emphatically that I am not one of those who decry their use. In drugs we possess a most valuable means for the diminution and often for the control of the seizures. I am of the opinion, based upon a not inconsiderable experience, that the harm done by drugs has been grossly exaggerated. The deterioration noted in an epileptic is properly to be ascribed not to the treatment or drug that he is receiving, but to the progress of the insidious disease from which he suffers. We must remember that epilepsy is not purely a paroxysmal disease; it is a continuous disease. The epileptic is an epileptic whether he suffers from convulsions or not, and in the interparoxysmal periods he is the same abnormal individual as regards both his morphology and his tissue-changes as he is during his epileptic attacks; in the latter it is merely that a crisis has been reached. Degeneration probably goes on in the

interparoxysmal periods as well as at the times of the seizures: though it appears that the frequent occurrence of the latter hastens the process. It is probably true also that the action of the bromides is less harmful than is the frequent occurrence of major attacks, and between two evils it is certainly wisest to choose the least.

The bromides, it will be remembered, were introduced by Laycock in 1853; their introduction constitutes, in my opinion, a veritable epoch in the history of therapeutics. To decry their usefulness is to deny the cumulative experience of almost sixty years. It is not, however, my intention to discuss the efficacy of bromides in epilepsy, but rather the important modification of the treatment introduced by Richet and Toulouse[11] in 1899.

We have all noted that there is in patients to whom we give bromides a tendency to bromide retention. This was long ago noted by Laudenheimer, who in studying a patient observed that of 80 grams of bromide administered only 35 grams were recovered from the urine. Of course some of the bromide must have been eliminated in other ways than by the kidney, yet the amount so eliminated must have been small. That there is a tendency to the retention of the bromides there can be no doubt, and this, as I have just stated, is in keeping with ordinary experience. Not infrequently, however, we fail to produce an adequate impression. The likelihood of such failure is greatly reduced if we institute the procedure of Richet and Toulouse, namely, that of chloride of sodium withdrawal. There seems to be an interrelation between the retention of the bromides and the elimination of the chlorides, and *vice versa*. Bromide accumulation occurs in proportion to the amount of the chloride of sodium withdrawn. Again, it would appear that the bromides are more readily eliminated after chloride of sodium is resumed. Ulrich[12] has shown experimentally that the elimination of the bromides in animals is enhanced by the giving of table salt. He also claims that the table salt rapidly removed the motor, sensory, and psychic phenomena of acute bromism. Bromide skin affections, he maintains, are rapidly dispersed by the administration of sodium

chloride. He regards table salt as an antidote for bromism. If to an an epileptic who is bromized and whose attacks have been controlled we give table salt, epileptic convulsions may recur. Frey[13] also has shown experimentally upon animals that there is a relationship between the elimination of the bromides and chlorides in the urine, and that a similar relationship can be demonstrated in the presence of these salts in the blood. The withdrawal of the sodium chloride always caused a retention of the sodium bromide. Bromide always acted more promptly when the chloride had been withdrawn. The resumption of the table salt by the animal at once hastened the elimination of the bromide. *Vice versa*, the administration of the bromide causes the amount of chloride in the urine to be increased. The kidney, Frey states, seems to make no distinction between the elimination of the bromine and chlorine. It would appear that an excess of either or both cannot be retained.

A great many observers have noted beneficial results from the withdrawal of the sodium chloride in the bromide treatment of epilepsy. Among these may be mentioned J. Voisin, R. Voisin, and Krantz.[14] Mirallié[15] has also had very favorable experience with this method. He believes that the withdrawal of table salt from the diet makes the nerve cells more sensitive to the action of the bromide. Lambranzi[16] concludes, after a very careful and thorough study, that the method results in the great majority of cases in a decided reduction in the number and intensity of the attacks and at times causes them to disappear entirely. He believes that the psychic condition of the epileptics is in the intervals of the seizures also beneficially influenced, and he points out that the extension of the treatment over a long time, for example three months, produces no unfavorable effect. Voisin and Krantz[17] weighed their patients and noted that there was at first, under salt deprivation, almost always a diminution in the weight of the patient, but that this loss of weight at once disappeared when the salt was resumed. The practical point resulting is that if in the persistent withdrawal of the salt we find a diminution of the body weight, this is to be looked upon as

an impairment of nutrition, which indicates a cessation of the salt deprivation treatment.

Kinberg[18] has published the results of thirty cases in which he employed the Richet-Toulouse method. He maintains (1) that the treatment lessens the number and intensity of the attacks and tends to prevent the recurrence of the status epilepticus; (2) that it often exerts a beneficial influence upon the mental state, and in this way prevents the occurrence of states of confusion and delirium. He believes that the method is contraindicated when there is disease of the heart, nephritis, emphysema, or adiposis; also if the patient loses steadily in weight the method is to be abandoned. Hoppe[19] points out that the bromides only become efficacious after they become retained, and dwells upon the importance of salt withdrawal. Muskens[20] treated in all 180 patients, among them 40 inveterate cases. Muskens believes that disease of the heart muscle constitutes a contraindication to salt withdrawal. Turner[21] out of eight cases noted a favorable result in five. In three this persisted for three months after a return to an ordinary sodium-chloride containing diet. In three cases he noted an increase in the number of petit mal attacks. He did not observe any improvement in the psychic symptoms. The chief disadvantage of the treatment he thinks lies in its monotony.

J. and R. Voisin and Rendu[22] recommend a modified method of the Richet-Toulouse method. They give the patients for short periods small doses of bromide, then for short periods large doses of the bromide, both with the usual salt-containing nourishment. Then these periods are followed by equal periods of salt-free diet but without the exhibition of the bromide salts. This modification does not appeal to me and I see no advantage in it.

Personally I have made use for some years of sodium chloride withdrawal and I share the opinions as to its value. The patient, as a rule, becomes rapidly accustomed to the absence of the salt. After a time some patients begin to crave the salt. If so, salt may for a short time be permitted, but later again withdrawn. If the prohibition of the salt be too radical, there may result a

marked loss of appetite and corresponding loss of weight. As in all other methods employed in diseases chronic and essentially incurable, the taking of the middle course, not too extreme in either direction, yields in the long run the best results. Measures such as the addition of bromides to the food itself, as, for instance, in the baking of bromide bread, so-called bromopan, I do not consider either necessary or advisable.

Other points in regard to the use of the bromides may be summarized as follows: (1) It is very important to use them early and in sufficient dose to control the seizures. It is a fact in accord with common experience that early in a case attacks can be more readily suppressed and controlled than later, and it should be our aim to prevent if possible by prompt and vigorous early measures the establishment of what may be called the convulsive habit, (2) the bromides should be given in sufficient dose and for a prolonged time. Gradually, especially in cases in which the general physiological plan of treatment already outlined is carried out, the dose may be diminished or even suspended for a time. We should bear in mind that the sudden leaving off of the bromide is unsafe.

An important point, I believe, in the administration of the bromides is to combine them with one of the glycerophosphate salts, preferably sodium glycerophosphate. There is an increased waste of phosphorus in epileptics, and some such procedure would seem to be indicated. In my own experience the combination of the sodium glycerophosphate has a beneficial action. Under no circumstances can it do any harm.

The Flechsig method of treatment hardly merits special consideration. It consists in the administration of opium in increasing doses at intervals during the day for a period of several weeks, five or six, after which time the opium is suddenly withdrawn and bromide given in full doses. There can be no doubt that in some cases in which the bromides fail to control the seizures, the Flechsig method proves efficient. I have on a few occasions, in bad cases, made use of it with satisfactory results for the time being.

Time will not permit of the discussion of the numerous other drugs employed in epilepsy. Of not one can it be said that they equal the bromides in value, and one only do I deem worthy of special mention, and that is thyroid extract. Thyroid extract appears to have a real though limited value. Browning, Osbourne, and Sajous have spoken of its use in suitable cases. As far as I am able to judge, it is in epileptic children with rather marked stigmata of arrest, with signs pointing to possible thyroid deficiency, that thyroid substance is valuable. It is occasionally valuable, however, in cases in which these stigmata are not pronounced. I think it extremely probable that its usefulness is to be ascribed to an increased oxidation of the tissues and a consequent destruction of toxic materials. It should be given in small doses over long periods of time. Large doses probably do harm—indeed, they may add to the frequency of the seizures. Beneficial effects cannot be looked for save after prolonged administration. The seizures should in the meantime be controlled by bromides; sooner or later in given cases it will be found that the dose of the bromide can be greatly diminished and even for a time suspended. An interesting point that possibly has some bearing upon the use of thyroid extract in the control or diminution of epileptic seizures and the possible role which increased oxidation plays in such a process, is the well-known clinical fact that in epileptics who suffer from a febrile infection, and in whom there is for the time being an increased oxidation of the tissues, no epileptic convulsions occur. It has been my own observation repeatedly that when an epileptic suffers from an attack of typhoid fever, for instance, or indeed from any of the other exanthemata, seizures are absent during the febrile period and frequently for some time thereafter.

Bearing upon the possible role of the internal secretions in the pathology of epilepsy, I have recently had skiagraphed a number of my epileptics with very interesting results in regard to the size and shape of the sella turcica. In several of the skiagraphs the sella turcica was shown to be greatly enlarged, leaving no doubt as to an increase in the size of the

pituitary body; others again revealed marked variations in size and conformation which appeared to be in excess of the variations found in normal individuals. These observations, necessarily incomplete, suggesting, however, very strongly disease of the pituitary in epilepsy, would justify the trial of pituitrin. Certainly in so distressing and unpromising an affection as epilepsy every clue should be followed.

I am entirely in accord with Dr. Mitchell as to the importance of determining the presence of an aura. Often too little stress is laid upon this fact, and yet it is a matter of long experience that a knowledge of the aura frequently enables us to abort the attacks by various expedients; of some of which Dr. Mitchell has spoken. I myself regard the aura as a sensory discharge which begins not in the periphery to which the sensation happens to be referred, but in a discharge which begins in a sensory area of the cortex; from such a sensory area the discharge travels either directly or by association fibers to a motor area, and arriving there a motor discharge, a convulsion, takes place. The question arises how does a sudden constriction of a limb, in which an aura is experienced, prevent the epileptic seizure? It is very probable that the constriction arrests nothing passing up the limb, but that instead it causes an impression to be sent to the brain into the corresponding sensory area of the limb, and for the time being arrests or changes the molecular movements which have started there and which would otherwise eventuate in a fully developed epileptic discharge. It is a not uncommon experience that a patient in whom an attack begins with an aura in a hand or finger can sometimes stop the attack by striking his hand or finger sharply against some solid object, such as a chair, table, or wall. No constriction is practised in such a case, and clearly a theory of a substituted sensory change is most in keeping with the facts.

Not only will the recognition of the aura permit of the use of various mechanical or physiological expedients, but it will also give us the opportunity of using amyl nitrite, a remedy first employed by Dr. Mitchell, which I fear we do not use as frequently

today as we used to, and which is certainly often efficacious in enabling us to abort attacks.

In regard to the arrest or abortion of attacks, especially by mechanical procedures, my experience has led me to the conclusion that this can be accomplished for a time only, and that the impulse or tendency to the convulsion sooner or later becomes so great that after a while all artificial barriers are broken down and the convulsion takes place. It would also seem that attacks checked by mechanical means or even by nitrite of amyl become cumulative—that is, when attacks finally occur, they are more severe and may occur in groups. Whether actual status epilepticus can be provoked under such conditions I do not know, but I would not deem it impossible. Again, as is well known, a patient in whom convulsions have for a long time been suppressed sometimes suffers from vague and often very distressing sensations —*e. g.*, depression, exhaustion, pressure sensations, about the head, fear, aching in the trunk, limbs, or abdomen. Occasionally the patient himself attributes these sensations to the fact of not having had a seizure. I remember one of my patients, in whom I was successful in suppressing the attacks for a long time by small doses of chloretone, telling me that she would much rather not take the remedy and have an attack than continue feeling so badly as she did. In certain cases an attack seems to be followed by an actual feeling of relief.

Together with Dr. Mitchell, I do not regard the symptoms which precede an attack sometimes for many hours as classifiable with the auræ. An aura is essentially and intrinsically a part of the epileptic attack. It is merely the sensory portion of the general cortical discharge. The preliminary symptoms of headache, depression, or other vague disagreeable sensations sometimes experienced by epileptics for several hours or a day before a seizure are possibly to be referred to an increased degree of toxicity, and are in their way analogous to the symptoms which sometimes precede an attack of migraine or in other patients an outburst of hysteria. They should suggest to us the use of a saline purge, perhaps a diminution of the food, or a more active administration of the bromide.

The toxicity of epilepsy has more than once suggested a serum method of treatment, but the failure of Ceni's attempt in this direction is so recent that it needs only to be mentioned. Attempts at the actual cure of epilepsy have thus far failed, and perhaps this is no reproach to our profession. We have to deal, it must be remembered, with an organism for the most part badly put together and intrinsically defective.

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THE SURGICAL TREATMENT OF EPILEPSY[1]

By WILLIAM J. TAYLOR, M.D.

ATTENDING SURGEON TO THE ORTHOPEDIC HOSPITAL AND INFIRMARY FOR NERVOUS DISEASES AND TO ST. AGNES' HOSPITAL; CONSULTING SURGEON TO THE WEST PHILADELPHIA HOSPITAL FOR WOMEN AND TO THE WOMAN'S HOSPITAL.

It is now nearly twenty-five years since I began assisting Dr. Keen in his cerebral surgery, and during these many years I have had the opportunity of seeing a vast number of cases of epilepsy and of operating myself upon a great many patients.

Epilepsies of every variety were brought to Dr. Keen, and for a time we hit almost every head that came in our way.

At first, and indeed at all times, the purely surgical results, the carpenter work if I may use such a term, were brilliant, so brilliant in fact that we became most enthusiastic and believed that in surgery the long looked for treatment of epilepsy had been found. As our experience ripened and sufficient time elapsed for us to see the real neurological results our enthusiasm waned, our disappointments were many, and finally we were compelled to modify our opinions and confine this method of treatment to a very limited number of carefully selected cases.

The needs for operation in traumatic epilepsy are now well recognized by all surgeons and neurologists and this is particularly so when there has been distinct injury to the skull producing a scar or depression of bone over the motor area, and when the seizures are focal in character.

It is quite generally believed also that in cases of injury producing persistent headache as well as in certain mental disturb-

[1] Read February 7, 1912.

ances an operation, even if only exploratory in character, is justifiable and at times imperative.

In the essential epilepsy of idiopathic or non-traumatic type where the attacks are general and without local onset no operation whatever should be undertaken. No permanent benefit can be expected, and it may and frequently does produce much harm.

Almost anything which is done to these patients may for a short time seem to modify the character and frequency of their attacks. Simple etherization, a fall, or even hitting them on the head with a club may apparently be beneficial.

Idiopathic epilepsy with focal symptoms, however, if seen early should always be operated upon, the dura incised and the surface of the brain examined carefully for evidences of cyst, new-growth, or localized meningeal inflammation.

When a focal epilepsy develops and is due to gross brain disease all surgeons are united in advising operation.

The earlier the operation the better the results, for after two years the associated fibers are almost certain to have undergone degeneration and little can then be promised.

The dura must be opened and the surface of the brain examined by sight and touch. If there be a fracture of the inner table of the skull or an overgrowth of bone, an organized blood clot, a tumor, a cyst, or other evidences of pathological changes such as adhesions between the bone or dura and the surface of the brain, efforts must be made to remove the source of irritation.

I do not think we are now justified in excising the centre from the convolution of the brain with any expectation of benefiting the patient, unless there be distinct evidence of disease.

If there be history of a trauma followed by epilepsy which has a focal origin even if the centre from which the impulse arises is not beneath the scar the surgeon should explore at the seat of the injury first and later over the discharging centre.

When the injury has been over the motor area the results from operative interference promise much more than when the injury is over the sensory region.

While the value of operations for epilepsy has been greatly exaggerated, and the results in the vast majority of cases disappointing, it does not follow that surgical intervention should be abandoned, but should be confined to carefully selected cases after the most thorough study by both neurologist and surgeon. Occasionally great benefit may result and unexpected pathological conditions found which admit of removal.

A wide opening should always be made in the skull generally by an osteoplastic flap, to enable the surface of the brain to be examined.

Dr. J. Chalmers Da Costa (*Medicine*, February, 1904), in an admirable article on the "Surgical Treatment of Epilepsy," gives a classification of this disease which is of the greatest value and one which I have studied with much profit.

PERSONAL OBSERVATIONS AND DEDUCTIONS AS TO THE PATHOGENESIS AND SURGICAL TREATMENT OF EPILEPSY, BASED UPON A SERIES OF SIXTY-THREE CASES[1]

By CHARLES H. FRAZIER, M.D.

To present a paper on the surgical treatment of epilepsy to a scientific body requires a word of apology, at least for the antiquity of the theme. I should hesitate to mention the date when surgeons first busied themselves with the problem, but it was a long while ago. And yet despite the many years of observation, the present status of surgical therapy in epilepsy is not altogether satisfactory, be it the fault of the surgeon, the neurologist, or the disease. So far as I know there is not to be found in surgical problems any quite analogous to that of epilepsy. Surgery is more or less of an exact science; pathology also is more or less of an exact science; when an organ is the seat of a lesion the pathological nature of which is thoroughly understood, and where the initial surgical experience would seem to indicate that that lesion may be favorably influenced by surgical therapy, we have a combination of facts which form a tentative basis upon which to formulate what appears to be a rational mode of treatment, and it is not long before the surgical profession proves the validity or the unreliability of earlier claims, and the operative treatment becomes a conventional mode of procedure subject to subsequent revisions or modifications or is altogether discarded.

[1] Read February 7, 1912.

The history of the surgical treatment of gastric and duodenal ulcer, of lesions of the biliary passages, as of many other lesions of the digestive tract, may be cited as an instance of the surgeon's invasion into a new field and of his gradual mastery of the problem by a steady but progessive movement in each step of which distinct advances are made. And one could cite innumerable instances of the extraordinary conquests in the surgery of the last two decades. But with epilepsy we must acknowledge at the outset a very checkered career. There have been waves of enthusiasm, followed by intervals of indifference, and at no time an entire unanimity of opinion as to the proper course of procedure. Were it not for the fact that we are dealing in most instances with an otherwise hopeless disease, and that at least in a small percentage of cases the patients have been altogether or partially relieved, surgeons would have abandoned the field long ago.

In the preparation of this paper at the outset I was somewhat skeptical as to the value of surgical treatment, and was agreeably surprised to find upon investigating my records that of 25 cases operated upon more than three years ago the results in 7, or 28 per cent., were more than satisfactory. Two of these were markedly improved, and five are virtual recoveries. These cases will be referred to more at length farther on.

To what should we attribute the rather chaotic situation as it exists today and the repeated failures to discover some mode of procedure which will receive universal recognition? To my way of thinking it is due very largely to our lack of precise knowledge of the nature of the disease with which we are dealing, and to the fact that the treatment whatever it may be has been worked out rather on old-fashioned, empirical lines than from an exact understanding of the true nature of the disease.

We have been told that in cases of so-called essential epilepsy there is no demonstrable lesion of the cortex either macroscopic or microscopic, and that in cases of focal or Jacksonian epilepsy there is frequently a focal lesion, but this is not altogether true —at least there are too many exceptions to warrant so positive a statement, for there are cases of epilepsy focal in character in

which the lesion is not focal, but diffuse in type, as well as those in which we fail to find any lesion whatsoever; and again there are cases of general or essential epilepsy in which we find lesions quite similar to those in focal epilepsy (see Fig. 1).

Inasmuch as every principle of successful surgical therapy is founded upon an intimate familiarity with the nature of the underlying lesion, it behooves us as surgeons to avail ourselves of every opportunity to make the closest observations of the

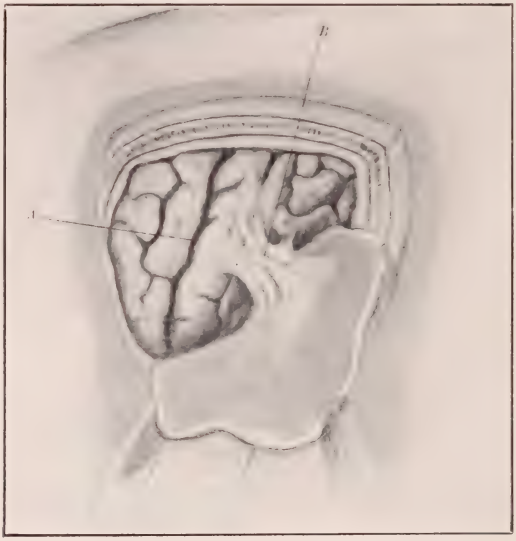

FIG. 1.—Reproduction of a sketch made at operation (File No. 3404), showing area of adhesions in postcentral convolution. This case illustrates a distinctly focal lesion with general convulsions. *A*, Rolandic fissure; *B*, focal lesion.

tissues exposed at operation. Of 53 operations upon epileptic subjects, which represent my surgical experience, in only 6 is it recorded that the findings were negative, and of the remaining the following conditions were noted: Cysts (8), adhesions (6), pachymeningitis with adhesions, edema or cysts (13), edema alone (11), with cyst (1), thickened dura (2), organized blood clot (1), cystic degeneration (2), cortical gliosis (2). I wish to call attention here to the multiplicity of the lesions with which we have

to deal and to the fact that a more or less definite pathological lesion was revealed in so large a percentage of cases.

Furthermore, the multiplicity of the lesions at once suggests the intricacy of the problem with which we are dealing, and the character of the lesions foretells the difficulty of devising means for their relief by surgical intervention. I do not want to dwell too much upon the pathogenesis, and yet it seems to me so vital to an intelligent discussion of the subject that one can ill afford not to give it a very conspicuous place. Of the above enumerated lesions, a considerable number, almost half, were classified as pachymeningitis or edema. As to the latter, we find in the subarachnoid spaces an unusual amount of cerebrospinal fluid, sometimes clear and sometimes cloudy like barley water, due to the presence of particles of lymph; upon pricking the pia and arachnoid with the point of the knife an unusual quantity of fluid escapes and may continue to drain for some time after the operation. I acknowledge the possible source of error in determining with precision at the operation whether the amount of fluid in the subarachnoid spaces is beyond the limitations of the normal, since a certain amount of fluid is present in these spaces under normal conditions. But there is no doubt in my mind, and this observation has been confirmed by many others, that in a not inconsiderable number there is a large enough collection of fluid to warrant the condition being regarded as different from what we are accustomed to see in the cortex of the normal brain. In a recent article, Alexander (*Lancet*, September 30, 1911) lays great emphasis upon edema as an important causation factor in the instability of the cortical cells to which epileptic seizures are ascribed. In 20 successive operations edema of the pia-arachnoid was recorded as present.

In addition to diffuse edema, I have indicated under the term pachymeningitis an opaque appearance of the membranes mostly confined to the course of the vessels in the sulci between the convolutions, but occasionally more diffuse, covering a portion of one or more convolutions. This opacity, I take it, is due to the deposition of lymph or to an exudate of traumatic or inflammatory

origin, as in not a few instances there has been a history of trauma or of some infectious disease, particularly scarlet fever. Whatever the origin or the nature, this pathological picture is often seen, sometimes alone, sometimes with adhesions, and sometimes with edema. I have made no reference to the microscopic pathology, as there has been no opportunity to make a histological study of the cortex in our clinical experience. Such changes as may be the result of congenital defects or of degenerative and inflammatory processes have been found by those who have made systematic examinations of epileptic brains.

Selections of Cases.—Of importance greater than the performance of the operation is the selection of cases. Because of its otherwise hopeless nature and because of the occasional recoveries following operation, the surgeon's advice and opinion are frequently sought, and in my experience the majority of cases present no surgical indications. I have never seen a case of so-called "reflex" epilepsy, but there is sufficient evidence from competent observers to satisfy me that sensitive peripheral lesions may be the determining factor in certain cases, and the propriety of operation under such circumstances should pass unchallenged. Reflex epilepsy as compared with other forms is infrequent. Traumatic epilepsy, on the other hand, is not unusual. In my series of 53 cases there was a history of cerebral trauma of more or less severity, with or without fracture in 27 instances. In almost every instance the patient or the parents will have remembered some incident in which an injury to the head was sustained, but a careful investigation should be made to eliminate those in which the injury or its consequences were comparatively trivial. It should be remembered, however, in this connection that seemingly trivial injuries in young children may be attended with hemorrhage or fractures, the presence of the latter being revealed by the radiograph (Denks and Schwarz, from Weil, *Beiträge zur klinische Chirurgie,* 1911, Bd. lxx). If there is no external evidence of injury, such as a cicatrix, a depression, or a cranial defect, a careful inquiry should be made as to what transpired immediately after the accident in order to determine

whether the clinical condition was serious enough to have been caused by a fracture, concussion, hemorrhage, or contusion (see Fig. 2); whether there was a period of unconsciousness, of delirium, of convulsions, of persistent headache, or of transitory paralysis of one side or the other. We are justified in recommending operation in traumatic epilepsy without external evidence of injury when the seat of the lesion can be surmised either from the nature of the attacks or from the symptoms attending the original injury. The seat of the lesion is clearly indicated in some cases by the character of the aura, and in others by the Jacksonian

FIG. 2.—A case of traumatic epilepsy with a large cicatrix and depression, the result of a compound fracture.

nature of the seizures. We are justified, I believe, in recommending operation in traumatic epilepsy in all cases in which there is external evidence of an injury, such as a depression of the cranium, more particularly defects of the cranium, but occasionally only cicatrices, where the history indicated serious cerebral trauma. The results following the removal of traumatic cysts, of fragments of bone, of thickened areas of dura justify the radical policy to which surgeons generally subscribe in their attitude toward traumatic epilepsy.

Much finer discrimination must be exercised, however, in

selecting cases from the group commonly known as symptomatic or general. In this group the convulsions may be Jacksonian or general. It is in the former, however, in which I have made it a practice to recommend an exploratory operation. In this group may be included the cases which follow the so-called cerebral palsies of children, in which the lesion found at operation may be the result of an encephalitis or meningo-encephalitis, such as porencephalus, cyst formation, gliosis, circumscribed granular plaques, or the pachymeningitis already alluded to. While in this group the character of the attack clearly indicates the area to be explored, the nature of the lesion is unfortunately not likely to be favorably influenced by any surgical procedure. That any operation could affect the gross extensive structural lesions that one sees in many of these cases is out of the question, so that as a class the epilepsies of cerebral palsies in my experience derive little, if any, benefit.

In the absence of residual paralyses or spasticities, one can determine the seat of the lesion in symptomatic epilepsy by a careful examination after the attacks, which may reveal some slight disturbance either motor or sensory in the extremities or in the distribution of the facial, trigeminal nerves (Krause), or nerves supplying the ocular muscles, or some disturbances of the reflexes. Thus by an examination after an attack or by the character of the aura (see Fig. 3) we may find a guide to the seat of the lesion even when the attacks are general and not Jacksonian, and under such circumstances I have been in the habit of advising exploration. In another group of cases, while the seizures are general at the time the patient comes under your observation, careful inquiry may reveal the fact that at the onset the convulsions were distinctly focal.

Briefly, then, I include in the indications for operation cases of traumatic epilepsy with such restrictions as have been indicated, cases of Jacksonian epilepsy, and cases of general epilepsy in which there are symptoms indicating a focal lesion. But no matter what the type, the operation must be undertaken with the full realization that the pathological lesion may be of such a character as to

defeat any attempt to arrest the disease. It is needless to say in this connection that there is nothing in the manifestation of the disease to indicate the nature or the extent of the lesion. Before the operation this is purely a matter of speculation; the most insignificant lesion may be associated with the gravest form of the disease, and *vice versa*, serious alterations in the structure of the cortex may give rise to attacks comparatively infrequent and relatively mild.

FIG. 3.—Photograph of patient at onset of general epileptic seizure, preceded by an aura of pain in the left leg, which the patient grasped firmly in the hope of averting an attack.

Operative Procedure.—The character of the operation must vary according to the character of the lesion, on the one hand, and perhaps the variety of epilepsy on the other. First of all as a matter of prophylaxis should we not be a little more painstaking in the examination of patients with cranial and intracranial injuries? If within a few days of the injury we can detect signs of hemorrhage, either epidural or subdural, the suspected region should be uncovered and the clot, if found, removed. I recall a case in which following a basal fracture and cerebral contusion I was able to detect slight weakness in the left upper extremity, and upon exploring the motor cortex I found and removed a small clot from over the arm centre (see Fig. 4). If

this had been allowed to remain, it could readily have become the exciting cause in disturbing the equilibrium of the cortex. In the line of prophylaxis also we should not only insist upon the elevation of fragments but upon the repair of defects following fractures of the cranium, if not immediately, at least as soon as the condition of the wound will allow. In justification of this I may quote from Gobiet (*Wiener klin. Woch.*, 1908, No. 21), who brings up the old question of the relation of cranial defect

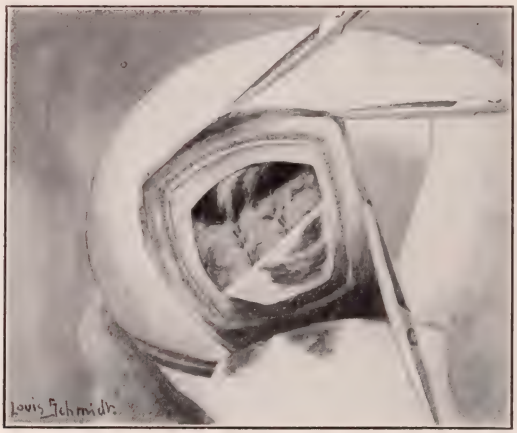

FIG. 4.—From a color photograph taken at operation, in which a thin pial clot was discovered in the region of the arm centre.

to epilepsy, and recalls Stieda's report of a series of thirty-three cases in which very favorable results were obtained by closing the defect.

In traumatic epilepsy with marked depression, the contour of the skull should be restored and at the same time the region beneath explored. Where we have large or small defects these should be repaired, preferably by the König-Müller method, transplanting a fragment composed of the outer table of the skull and pericranium to the defect and securing the same in place with interrupted sutures (see Fig. 5). While I have in the past used celluloid plates, I have abandoned them altogether for the autoplastic method, and have now under observation four cases,

none of which have as yet, however, passed the three-year limit. Adhesions are just as big a bugbear in the cranial as they are in the peritoneal cavity. When they form, as they frequently do between the dura and the pia-arachnoid, either they should be left undisturbed or we should be prepared to sacrifice a portion of the dura involved, especially if it overlies that region of the cortex from which the convulsions are initiated. The mere division of adhesions will be followed inevitably by their reformation, and the interposition of foreign material is impracticable, as the latter acts as a foreign body and source of irritation.

FIG. 5.—Photograph of patient with traumatic epilepsy, with large cranial defect.

There need be no discussion as to the management of cysts. The latter should be evacuated and as much as possible of the cyst wall removed, but if the cystic formation is merely a part of a porencephalic process or of cystic degeneration, the mere evacuation or removal of the cyst will have little if any influence upon the epileptic attacks (see Fig. 6). The same general principle applies to all focal lesions as applies to cysts. No matter what their nature, whether tumors, gumma, organized blood clots, fragments of bone, thickened dura, or what not, they should be removed. Thus far the problem is comparatively free from difficulties; when, however, we are confronted with cases in which

the process is more diffuse, as in the cases of edema or in those in which there is no demonstrable lesion, we must choose one of several methods: (1) A method designed to afford drainage, (2) excision of the cortical centre, Horsley's method, and (3) the decompression method of Kocher, the so-called "*ventilbildung.*" Having had no experience with Bircher's method, I will pass it by without comment; suffice it to say that Bircher recently recommended gentle massage of the cerebral cortex and believes his cases were favorably influenced by the operation.

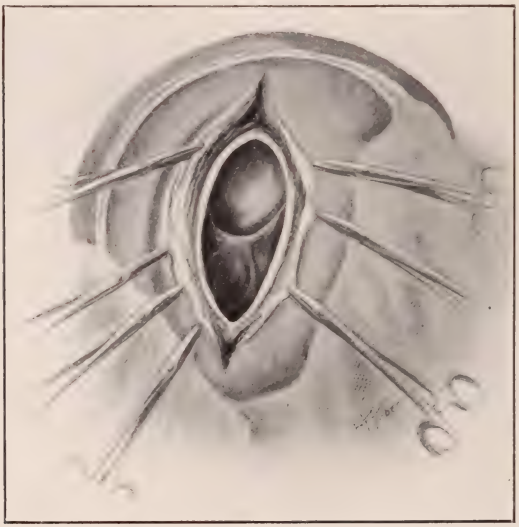

FIG. 6.—From a sketch, made at operation, showing extensive area of cystic degeneration of traumatic origin.

In dealing with cases of edema alone, I have been in the habit of pricking the membranes at various places in order to allow the fluid to drain off and of providing some outlet to the epidural space either by having a portion of the dura unsutured, by removing a portion of the dura (see Fig. 7), or by a method about to be described, which commends itself especially to me, suggested by Krause. After the osteoplastic flap is reflected, an incision is made in the dura one-third to one-half an inch from the margin

on three sides of the cranial opening. The dural flap is reflected, and at the four angles of the opening the dura is cut up to the margin of the bone. This enables one to reflect three dural flaps, so that when the osteoplastic flap is replaced a layer of dura is so interposed between the bony surfaces as to prevent their union. If we recognize in edema a pathogenic factor, and believe the convulsions will be favorably influenced by drainage, I know of no better way in which drainage can be provided for than by the method as above outlined. In looking over my records, I find

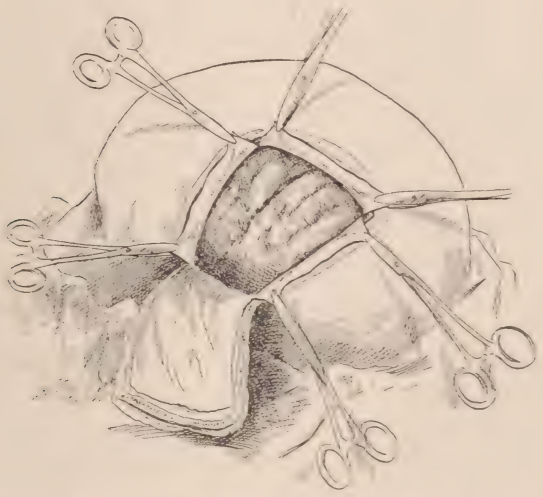

FIG. 7.—Schematic representation of method of providing drainage in cases of edema. Note narrow marginal dural flaps reflected over margins of bony opening.

that in 23 of 53 operations I have concluded the operation by removing the bone from the temporal region as for a cerebral decompression. Many of these cases are too recent (7) to include in the final report, but of the older cases (16) there is one who in over three years has had but one mild attack, a virtual recovery.

When dealing with cases with focal symptoms, but without focal lesions, we must adopt one of two methods, Kocher's decompression or Horsley's excision of the cortical centre. As for the former, although not convinced of the rationale of the operation,

I have practised it a number of times, especially within the last three years, because it has been recommended by so eminent an authority as Kocher, and received the indorsement and the favorable results reported by Friedrich, Kümmel, Krause, and Kocher himself. Kocher proposed decompression on the theory that epileptic seizures are provoked by a condition of intracranial hypertension. This idea has been confirmed by observations of Hause and Hitzig, and of Stadelman, while, on the other hand, Nawratzki and Arndt look upon increased tension as an effect rather than a cause.

With Horsley's operation my experience has been limited to one case in which the facial centre was excised without any appreciable benefit. This method in its day was enthusiastically received, later was practically abandoned, and more recently has been revived. The failure attending its practice has been attributed to the fact that surgeons determined the area to be excised only by anatomical guides rather than by the exact methods of faradization. My own objection to the operation was based on the theory that the resulting scar would in itself be sufficient to excite convulsions and that recurrence would be the rule rather than the exception. If further observations do not sustain this argument, I should be inclined to adopt it in those cases in which no other lesion is found and in which the centre can be accurately determined by faradization.

Results. Extraordinary reports have been issued from time to time as to the effects of surgical intervention, extraordinary chiefly in the great variability in the percentage of recoveries in the hands of different operators. It is difficult to account for this great variation, unless it be that in many instances the reports are issued too soon after the operation. To standardize the result, no case should be reported as cured unless at least three years have elapsed since the operation. Of course the three-year limit is purely arbitrary, although quite reasonable, as relapses or recurrence after that time are exceptional, but the well-known effect of operation *per se* upon epileptic subjects makes it imperative that a reasonably long time should have

elapsed before any case should be put on record as influenced by the operation. The effect of operation *per se* upon epileptic subjects has never been satisfactorily explained, and yet no one who has had the opportunity of observing many cases in the postoperative period could fail to recognize the change of temperament, of general morale, as well as the freedom from attacks. Often have I had patients tell me in the convalescent period that they had not felt so well for five or ten years. We cannot attribute the transformation altogether to the relief from attacks, for in many cases the attacks may have been comparatively infrequent. Can it be due to any modification of the metabolic processes or to any influence upon glandular secretions, to change of environments, of habit, or of diet, to any factors which may influence the nutritive process of the body in general and the brain in particular? We can only conjecture as to this, but the question has arisen in my mind as to whether the effect of operation *per se* may in some cases be more than transitory in effect and account for a certain number of those cases which have been reported as cured irrespective of what particular technique may have been carried out. In view of the doubtful efficacy of some of the operative procedures which have been practised, I cannot help but feel that there is some foundation for this hypothesis.

Turning now to my own records, out of 25 cases which have passed the three-year limit and could be traced, 7, or 28 per cent., have been profoundly influenced by the operation. Of these 7, one (File No. 982) was a case of Jacksonian epilepsy of five months' duration, in which a subcortical cyst was found at the operation and the cyst wall removed. He recovered considerable strength in the arm and leg, which were partially paralyzed, the headaches of which he complained were entirely relieved, and his attacks were reduced in frequency to one every two months and are much less severe than they were before.

In the second case (File No. 1102) the pathological lesions were a pachymeningitis, edema, and increased tension. The operation was performed six years ago; the attacks, of one year's duration, at first were of the Jacksonian type and afterward became gen-

eral; there have been no attacks for the last three years; the patient, now a young man of nineteen years, is of sound physique, able to work, and his mind is much clearer than it had been.

The third patient (File No. 1249) was a young man of twenty; since the operation in 1906 the attacks have become progressively less severe and less frequent; he has been entirely free from attacks for the last nine months. A diffuse pachymeningitis and cortical cyst were uncovered at the operation. The patient's disposition has improved, he is able to work, and in every other respect he is perfectly well.

The fourth patient (File No. 2482) within a year of the operation in 1907 had sustained a serious cerebral contusion without focal symptoms, but with loss of consciousness followed by delirium extending over a period of several weeks, and an organized blood clot was found in the motor region and removed. At the site of the clot therewere firm adhesions between the dura and pia. Four years have elapsed since the operation, and in this time there have been no attacks for the last three and a half years. This case emphasizes the importance of operating upon all cases of cranial trauma in which there is any reason to believe there has been a cortical hemorrhage. Had we been able to detect any focal symptoms at the time of the injury we would have urged operation without further delay, but despite frequent and careful examination no evidence of cortical hemorrhage was apparent.

In the fifth of the series (File No. 2683) nothing was found at the operation but an intense edema; although the patient had sustained a fracture of the skull there was no evidence of any injury to the internal table or to the underlying meninges and cortex. Before the operation he was having two or three attacks a week, all general in character, but he has had none since.

In the sixth case (File No. 3404) the patient was twenty-three years old at the time of the operation, three years and nine months ago. She had had concussion of the brain at eleven, her first convulsion at fifteen; the attacks were general in character. An area of adhesions two centimeters in diameter was found a little

behind the postcentral convolution. Although almost four years have elapsed, the patient has had but one attack, of which she had no intimation, as it was not severe and occurred at night.

In the seventh case (File No. 3626) the patient, a young man of twenty, had sustained a fracture of the skull at the age of six. The operation, performed almost four years ago, revealed an intense edema and a pachymeningitis, and since that time he has had no convulsive seizures. He has been in splendid health and able to attend his work regularly during the four years intervening.

Briefly, then, of 25 cases operated upon more than three years ago, 7 have been profoundly influenced:

SUMMARY

Case.	Date of operation.	Character of attacks.	Duration of disease.	Findings.	Operation.	Result.
1	1905	Jacksonian.	5 months.	Subcortical cyst.	Evacuation of cyst.	Attacks less frequent and less severe—once in two months.
2	1905	General.	1 year	Pachymeningitis and edema	Craniotomy	No attack in three years.
3	1906	Jacksonian	6 years	Pachymeningitis and cyst	Evacuation of cyst	Gradual subsidence of attacks; none for last nine months
4	1907	General	6 months	Organized blood clot	Clot removed.	Only one since operation; none for last 3½ years.
5	1907	General	?	Edema	Craniotomy	Has had no attacks since operation
6	1908	General	8 years	Adhesions and pachymeningitis	Craniotomy and decompression	Only one mild attack in four years
7	1908	General	14 years	Edema and pachymeningitis	Craniotomy and decompression	Has had no convulsions or seizures since operation

These results are by no means brilliant; but they are sufficiently gratifying to stimulate further investigations and renewed efforts. In his report to the German Surgical Congress in 1910, Tillmann presented a series of 29 recoveries beyond the three-year limit. In Krause's personal experience (*Allgemeine wiener medizinische*

Zeitung, 1910, iv) with 50 cases there were 5 completely cured or greatly improved. These figures correspond quite closely with my own as to the total number of cases and the number of cases benefited by operation. The value or the justification of the operation should not be measured by the percentage of absolute cures alone. To reduce the attacks in severity and in frequency from one in several days or weeks to one in several months or years, or even to be able to arrest the progress of the disease, is a matter of no small consequence and should not be last sight of in the final analysis.

It has not been my purpose to advance the claims of surgery as a means of controlling a very distressing disease, but merely to present such observations as I have been able to make at the operating table in the hope that they may throw some side-light upon a problem as peripatetic as it is perplexing.

SUMMARY

1. The development of the surgical treatment of epilepsy has met with many impediments, among which should be mentioned: (*a*) The uncertainty as to the origin of the attacks; (*b*) the multiplicity of the pathological lesions; (*c*) the positive findings in some cases and negative findings in others.

2. In a larger percentage of cases than has been hitherto appreciated, some pathological lesion of the exposed cortex has been found.

3. Operative intervention should be regarded as justifiable: (*a*) In traumatic epilepsy with external evidence of an injury; (*b*) in traumatic epilepsy without external evidence of an injury when the nature of the attacks or the symptoms immediately following the injury indicate the seat of the lesion; (*c*) in all forms of Jacksonian epilepsy of whatever origin; (*d*) in general epilepsy where the suggestion of a focal lesion may be found by a careful physical examination before or after the attacks in some disturbance of motion, sensation, or reflexes.

4. The operative procedure must be adapted to the character of the lesion: (*a*) Cranial defects should be repaired; (*b*) focal lesions, such as cysts, tumors, etc., should be removed; (*c*) edema, a very common lesion, calls for some method of drainage; (*d*) in Jacksonian cases without lesion the cortical centre should be excised; (*e*) in idiopathic epilepsy without focal symptoms or lesions Kocher's decompression deserves consideration.

5. The value of the operation may be measured in terms of improvement or cure. Either should be considered as justifying the operation.

6. Gratifying results may be anticipated in at least 10 to 25 per cent. of cases.

STATISTICAL SUMMARY

1. Number of Cases	63
2. Number of Operations	53
3. Character of Operations:	
Craniotomy	21
Decompression	3
Craniotomy and decompression	20
König-Müller repair of defect	4
Evacuation of cyst	3
Clot removed	1
Excision of facial centre	1
4. Sex:	
Males	41
Females	12
5. Ages According to Decades:	
0–10	7
10–20	16
20–30	18
30–40	10
40–50	2
6. Etiology:	
Fracture	9
Cranial trauma	18
Intracranial hemorrhage (newborn)	4
Epidural hemorrhage	1
Cerebral hemorrhage	1
Tumor	1
Chronic meningitis	1
Infectious fevers	8
Undetermined	10

7. Character of Attacks:
   - General . . . 31
   - Jacksonian . . . 18
   - Jacksonian and general . . . 4
8. Duration of Disease:
   - Less than 6 months . . . 10
   - 6 months to 1 year . . . 3
   - 1 year to 2 years . . . 8
   - 3 years . . . 7
   - 4 years . . . 2
   - 5 years . . . 5
   - 6 years . . . 4
   - 7 years . . . 1
   - 8 years . . . 4
   - 9 years . . . 1
   - 10 years . . . 1
   - 11 years . . . 1
   - 15 years . . . 1
   - 20 years . . . 1
   - Duration unknown . . . 4
9. Findings at Operation:
   - Cyst . . . 8
   - Adhesions . . . 6
   - Adhesions and pachymeningitis . . . 2
   - Edema . . . 11
   - Edema and pachymeningitis . . . 8
   - Dura thickened . . . 2
   - Organized blood clot . . . 1
   - Cyst and pachymengitis . . . 1
   - Cyst and edema . . . 1
   - Cystic degeneration . . . 2
   - Softening . . . 2
   - Pachymeningitis . . . 2
   - Negative findings . . . 6

DISCUSSION

Dr. Hobart A. Hare: Dr. Mitchell has not alluded to the point to which he was the first to draw attention, that many cases of minor epilepsy are differentiated by virtue of the fact that inhalations of nitrite of amyl make them worse rather than better. To a certain extent this is a therapeutic diagnostic fact.

The impression I have received from this discussion in its entirety is that, while we have no clue to the instability of motor or sensory nerve cells, we must recognize that epilepsy as we know it, is not a definite disease any more than is diabetes mellitus, but rather some condition of instability of nervous matter which can be set off by myriads of causes. To those of us who followed the investigations made by Albertoni many years ago, in which he controlled by bromides convulsions produced by the faradic current applied to the cortex, it would seem that there is a direct influence of the bromides upon the excitability of nerve cell. Such a condition may be dependent upon some alteration of the circulation or blood-vessels in the brain as Dr. Mitchell has suggested. Then, too, we see in eclamptic women true convulsions with a toxic cause. The fact, too, that brain tumors and fractures of the skull result in these convulsions indicates that this eruptive condition of the nervous system can be brought into play by causes as far apart from those which apparently produce idiopathic epilepsy as can possibly be.

Some years ago I had opportunity to confirm the view expressed by Dr. Mitchell and Dr. Shanahan concerning the prophylactic value of nitrite of amyl in cases which have an aura, and became impressed with the fact that it had the power to diminish the number of attacks.

Regarding the withdrawal of salt from these patients, until tonight I had presumed that the idea of taking away the salt was that the tissues would be so starved that they would actively take up the bromides.

Dr. Charles K. Mills: Regarding the medical treatment of epilepsy I have but little to say beyond that which Dr. Mitchell has said. In my own experience sodium bromide and lithium bromide have been the remedies which seem to me to produce the best results alone or in combination. I have found in many cases that combinations of the bromides with small doses of arsenic and conium will give excellent results, so far as these can be obtained from medicines. Little has been said about regulating the time and method of gradual reduction of the bromides. This is important. The first thing is to secure the dose which will diminish, or for a considerable time, prevent the attacks; the next is to determine the time at which the bromide mixture should be administered, and the interval of omission of the bromides which should be allowed after a certain time of administration has elapsed. This, of course, can only be reached by some empirical method and must be based upon personal experience with every case.

One thing that has been overlooked I think is that Jacksonian epilepsy is often a manifestation of general or so-called idiopathic epilepsy. In my early experience with operations I overlooked this now and then myself.

There may be a Jacksonian epilepsy inside, so to speak, of the general

epileptic attack. This has sometimes led the neurologist and the surgeon to a belief in the presence of a localized gross lesion in the motor zone.

With regard to epilepsy and pituitary disease I recall three cases with spasms and such uncinate symptoms as the dreamy state, taste or smell aura, smacking of the lips, etc., in which I had *x*-ray investigations made of the region of the sella turcica. In all of these cases more or less disappearance of the posterior clinoid process and other changes in the sella turcica had taken place. These were cases apparently either of tumor or other lesion of the pituitary body or growths of the uncinate region invading the sella turcica. In these cases the convulsions seem to be due either to the tumor invading the pituitary region or to disease of the pituitary body itself. Operations have been performed by tapping the sella turcica from below or by approaching this region by way of the anterior portion of the middle fossa. While such operations are difficult, it is not impossible to make use of them successfully either by drainage of the pituitary through the base of the sella turcica or by removing the growth in whole or in part when this is isolated. I have become interested also in the thyroid pituitary treatment of epilepsy, which I have used to a limited extent, but I have not as yet sufficient data upon which to base conclusions.

Dr. F. X. Dercum: As regards the pituitary body, one of my cases was most interesting. It was that of a man, who at about the age of thirty years began to be suddenly awakened at night with a sensation of a horrible odor which he could not describe. There were no other symptoms—no dream, no convulsive seizure. After these attacks had persisted at varying intervals for some ten years, they began to be attended by convulsive phenomena until a typical picture of nocturnal epilepsy resulted. Seizures at length also occurred during the day. The skiagraph in his case revealed an enormous enlargement of the pituitary fossa with complete disappearance of the posterior clinoid processes. An examination of his person also revealed distinct acromegalic changes, though these were not pronounced.

It has never been my experience to have a patient have a convulsion during a clinical lecture and I have but rarely had a patient have a fit in the waiting room of my out-patient department. I have had but 2 cases with attacks in my office. One man who had essential epilepsy had an attack just as he was leaving and the other was a case of Jacksonian epilepsy. It is a fact, in my experience, when epileptics are mentally interested by unusual surroundings, such as the clinic room, the attacks are inhibited. I should like to ask Dr. Mitchell what his experience has been in that respect.

Dr. S. Weir Mitchell: My experience has been rather the reverse of that of Dr. Dercum. During the Civil War I had a ward of epileptics, and

when making my morning visit it was quite a common thing to find four or five men go off in attacks. It was also a favorable time for men who were simulating epilepsy.

I do not agree with my friends upon the tremendous necessity for regulating the hours, diet, exercise, and everything else of confirmed epileptics. I can put this better by illustration: A gentleman consulted me a few years ago who said: "I have a statement of how I am to live. I have 15 or 16 attacks in a year. If I do not follow this schedule, which is very unpleasant, how many more will I have in the year? I know I can control the attacks moderately with bromides. If I take more bromides I am made unhappy. I can take a certain amount of bromide, but here is all the rest of this misery. I have tried to follow this schedule for three years. For one of those years I followed it rigidly. In the second year I did everything I pleased and had fewer attacks." I think it is undesirable to tie these patients down to rigidity in all sorts of directions.

THE TREATMENT OF SACCULATED ANEURYSM BY WIRING AND ELECTROLYSIS[1]

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The treatment of sacculated aneurysm by the introduction of various foreign bodies, ranging from nails to strands of silk and horse-hairs, was practised many years ago. The design was of course to aid or produce clotting in the sac and so to reënforce the weakening aneurysmal wall. These measures failed chiefly because most of the operations were performed before the days of asepsis, or else they caused so much damage to the sac wall that its continuity was destroyed and the end hastened. It is not my intention in this paper to go into an historical review of these cases, or of the early trials of more recent methods, such as the Moore-Corradi operation, which I have performed on twenty-two occasions[2] for saccular aneurysm of the various parts of the aortic arch. Six of my cases are now recorded for the first time. In every instance the growth was so large that death in the early future was imminent, and in several the wall of the sac was so thin as to be blue or blackish in hue like a great ecchymosis, and bloody serum was oozing through the skin.[3] In most of the cases death because of the aneurysm eventually took place, but in many, if not all, life was prolonged, so far as human judgment can determine, and equally important, there has been practically in

[1] Read March 6, 1912.

[2] Since this paper was written I have operated on two other cases.

[3] Pictures of two of these patients are to be found in Therap. Gaz., January 15, 1900; of another, January 15, 1903.

all instances relief, or partial relief, from pain which was severe before the operation.

Much depends on the general state of the aortic wall. If the aneurysm is solely the result of trauma and the rest of the vessel wall is healthy it can withstand the pressure of the blood stream. Thus a patient reported by my chief of clinic, Dr. Beardsley, is now in my wards,[1] twenty-seven months after operation, without any disablement, dyspnea, dysphagia, or pain, and with no sign of aneurysm save a heaving impulse at the second and third left

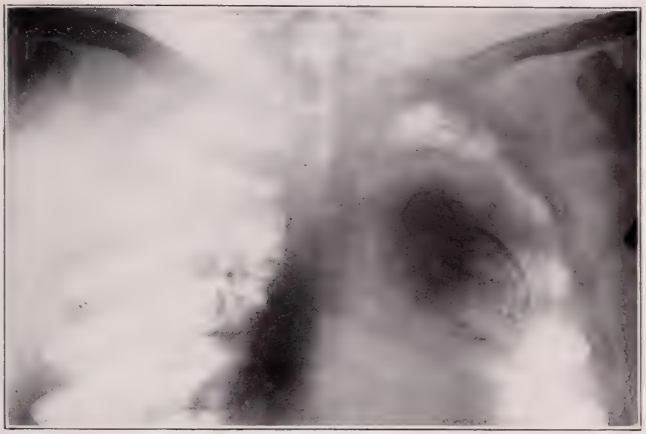

FIG. 1.—Aneurysm of the aorta, showing wire in the aneurysmal sac, two years after operation, with total arrest and no discomfort: from a skiagraph. (Case originally reported by Dr. Beardsley in the Therapeutic Gazette, March 15, 1911.) Case VI.

interspace in the midclavicular line. The growth has been entirely controlled. The aneurysmal sac in this case with the wire coiled in the clot is shown in Fig. 1. In a patient (operated on by the late D. D. Stewart) who was under my care for many months, although the growth projected nearly three inches from the chest, the patient lived in perfect health for three years, and died not from his aneurysm, but from pneumonia due to an alcoholic debauch. In this case the autopsy revealed the wire so firmly

[1] He is kept there for demonstration purposes.

embedded in organized clot that it could not be removed except by corrosion methods.

On the other hand, if the vessel wall be severely and generally diseased the proposition is akin to an attempt to mend a piece of rotten rubber hose. Some time after it is supported in one spot it gives way at another point. If the second point of yielding is so placed that it can be recognized and reached by the hollow needle used to insert the wire, it also can be supported, and in four cases I have done a second operation, and in one case a third, thereby preventing rupture at these points. In the latter case a fourth soft spot on the posterior wall of the sac gave way, and six

FIG. 2.—Aneurysm a few hours after wiring. The mass was less expansile and firmer than before.

months later caused death. Yet in this case the skin at each point of wiring when operated on was so thin as to be weeping bloody serum. The giving way of adjacent tissue when the first point of weakness had been supported is illustrated in Figs. 2, 3 and 4.

While it may be justly asserted that the number of cases in which life is greatly prolonged is very limited, on the other hand, it must be borne in mind that the condition is, except in the rarest instances, invariably fatal and cannot be materially benefited by any other measures. Further, the condition is often unrecognized until the growth is very large and the patient's state has become desperate. Thus in the cases now reported for

the first time the operation was refused until the symptoms grew so desperate that the patient was willing to resort to any measure.

FIG. 3.—Same patient as in Fig. 2, showing how, the upper area having been consolidated by wiring and electrolysis, the sac developed a second point of yielding five days later.

In one of them pulmonary edema was already well developed as a result of pressure and the patient died four days later of this condition, with the gain, however, of great relief from terrible

FIG. 4.—Same patient as in Figs. 2 and 3, thirty-eight days later. The spot at which rupture threatened when operation was done is still firm, but the underlying growth has grown enormously.

pain from which he had suffered for weeks. The pictures of this patient showing the tumor of the chest during life, and of

the sac obtained at autopsy with the wire coiled in it, are seen in Figs. 5 and 6. In another case operation was refused until after a lapse of two months the growth had tripled in size, large doses of morphine were needed to relieve the great pain, partial aphonia had developed, and expansible pulsation had extended for 6 inches down the vertebral column on the left side from the site of the original sac (Fig. 7). Here again the operation was performed as a last resort, with small hope of cure, and chiefly with the belief that the pain would decrease, which it did. Death took

FIG. 5.—Aneurysm of the ascending arch of the aorta a few hours after wiring electrolysis.

place twenty-eight days later from pulmonary edema, a not unexpected result, as the left lung was already impaired by pressure at the time when the wiring was performed.

In another of these cases the patient absolutely refused to remain in bed, getting up several times night and day, beginning this breach of orders immediately after the operation, and thereby preventing the hardening and fixation of the clot about the wire. In still another case, in which the growth had eroded the chest wall and was very prominent,[1] through the error of an assistant

[1] Therap. Gaz., Jan. 15, 1903.

the non-insulated shank of the needle came in contact with the skin, already distended and devitalized, and caused an electrolytic

FIG. 6.—Saccular aortic aneurysm, showing wire coiled in the sac. The specimen is split vertically and transversely and turned back. Most of the clot has been removed to show the wire. In the median line is seen the small opening in the aortic wall which communicated with the sac. From the patient in Fig. 5.

burn. This never healed; the slough extended and so weakened the sac wall that death ensued because the clot was pushed out of the sac through this orifice. This result, however, did not take place for months, during which time the patient was well enough to travel and to get married.

The six unreported cases are as follows:

Case I.—A negro, aged forty-three years, presented a large diffuse swelling in the midsternal line extending into the episternal notch and downward to a point four inches below. Laterally the growth extended two and a half inches to each side of the median line. The sterno-clavicular joint on the right side was dislocated. The growth did not give a bruit, an expansile pulsation, or a thrill. After excluding sarcoma by the aid of the *x*-ray, the operation was advised and refused. Three weeks later the patient returned with marked dyspnea, incessant cough night and day, and the signs of beginning bilateral edema. At this time the growth was far larger than before, and the patient was very hoarse. Two days were lost getting the needful wire. Fig. 5 shows the patient just after the operation. Thirty-five feet of gold wire were passed into the sac through an insulated needle. Beginning with 5 milliampères, the current was increased by 5 additional milliampères every five minutes until 50 milliampères were passing, and then the current was gradually turned off. During the operation the patient, although forced to lie in an uncomfortable posture, expressed himself as largely relieved of pain without being prompted to do so by a question as to suffering. The growth also felt firmer and harder at the end of twenty minutes. For a few hours after the operation the cough decreased, and the pain was greatly diminished until death took place from pulmonary edema four days later. The result of the autopsy was the confirmation of the diagnosis of bilateral pulmonary edema, and both vagi were found to be involved in the growth. The aneurysmal sac with the wire coiled in it and nearly all the clot removed is seen in Fig. 6.

Case II.—A white man, aged thirty-eight years, giving a long history of severe pain in the left arm, which had been diagnosticated as neuritis and treated with salicylates and heat, was found to have an aneurysm which had its greatest point of expansion posteriorly at the spot marked on Fig. 7. There was a slight bruit and distinct heaving impulse at this point. The *x*-ray revealed a sacculated aneurysm of the latter part of the transverse and beginning of the descending aortic arch. There were no additional symptoms except severe pain. Here again operation was refused for two months and then called for as a last resort after the growth had greatly extended. The radial pulse in the left wrist was at

that time almost, if not quite, absent. The left lung was dull on percussion along its posterior border in both lobes and the heaving impulse reached from the growth to the level of the sixth interspace in the paravertebral line. The needle was inserted at the point marked in Fig. 7, and 35 feet of gold wire introduced. Here again the patient, who was groaning with pain when the operation began, volunteered the information that the

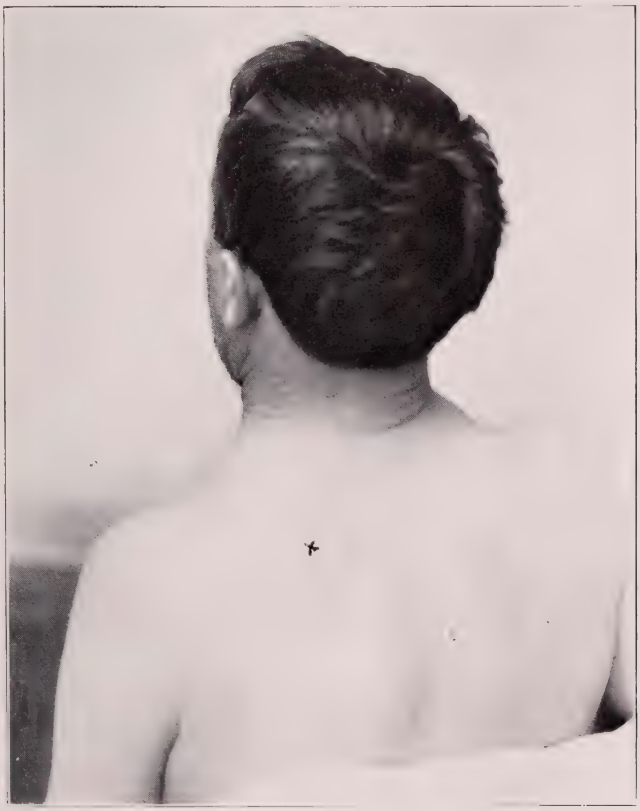

FIG. 7.—Aneurysm of the transverse and descending arch. Cross indicates point at which greatest heaving and expansile impulse were noticed and where needle and wire were introduced. Case II.

pain was less after the current had been passing for twenty minutes, and a little later, while the operation was being continued, he spoke jokingly about political affairs. The growth was now found to be distinctly firmer Death followed from pulmonary edema in twenty-eight days. In this case the pain was decreased but not stopped, probably because the growth

had already damaged the vertebræ in its downward progress. No autopsy was obtainable.

Case III.—W. K., aged forty-four years, had a brother whose death was probably due to aneurysm, as he died immediately after feeling something burst within him. The aneurysm in this patient arose near the sternum on the right side at the third rib.

An *x*-ray examination showed a saccular aneurysm of the ascending arch. The pupil on the right side was larger than on the left. In the upper part of the chest to the right of the sternum was a distinctly visible pulsating area about 4 inches in diameter in every direction. The apex beat was in the seventh interspace in the left anterior axillary line. There was a systolic bruit over the aneurysmal sac, but no difference between the radial pulses.

Ten feet of gold wire were introduced at 12.45 P.M. After the introduction of the wire the operation was delayed for half an hour because of trouble with the milliampèremeter. The current was gradually increased 5 milliampères every five minutes until 50 were passed, when it was gradually diminished until it was stopped. Eight minutes after the current began passing the needle was noticed to be moving less than before, and the growth when palpated was firmer. That night the patient said that he felt the wire in his chest and asked for something to make him sleep, as he felt nervous. He complained of backache, which he thought was due to the position he had occupied on the operating table. The next day he felt better. It was impossible to keep him in bed, and he disobeyed orders continually, arising and going about. Thirty-seven days after the operation the patient seemed to be still improving, but had some discomfort because of the bandage across his chest. The following day he complained of violent pain in his right side. The respirations became rapid and shallow, the lips and the face pallid. At 5.50 P.M. the patient was seized with an attack of tumultuous heart action, with staring eyes, and death occurred in twenty minutes on the thirty-eighth day after the operation. In the cavity of the tumor was found the wire which had been introduced. The right pleural cavity contained 1900 c.c. of blood clot and 1600 c.c. of serum, the sac having ruptured laterally into this space.

Case IV.—A man, aged fifty-four years, had had pain under the sternum for several years. Six months before examination he had noticed a swelling coming from under the sternum which had eroded this bone and the second, third, and fourth ribs on the right side and projected about one inch above the chest-wall. He attributed his difficulty to a kick on the chest by a pack-mule four years before. This patient was operated on on two occasions at an interval of a month and died eight weeks after the first operation and two weeks after the second, pressure on adjacent

tissues causing pulmonary edema. He willed his body to me, and with the intention of having frozen sections made of the torso during the summer, it was placed in a large jar. No trace of specimen or jar could be found later, and it is supposed that some laboratory servant broke the jar and destroyed the specimen in order to avoid censure. The complete clinical notes were also lost.

Case V.—A white man, aged forty years, had a large diffuse heaving tumor to the left of the sternum from the second to the fifth rib. There was expansile pulsation and a bruit. The growth developed some months after the patient had been thrown from a car, striking his chest on a stone. When told of the operation as a measure of relief he asked that it be done at once, as the pain was so severe that he would kill himself rather than submit to it any longer. There was also urgent dyspnea. Before the electricity had been passing fifteen minutes the patient volunteered the statement that his pain was much relieved. He was warned of the need of rest, and after a month in bed took a journey of several hundred miles. He transacted a large business for several months lying on a couch, and then began going about. Two years and a half after the operation he returned and the skiagraph showed complete arrest of the growth. Six weeks later he suffered from a severe attack of influenza, developed pulmonary edema, and died a little over two years and eight months after the operation. No autopsy was obtainable.

Case VI.—This case has already been reported by Dr. Beardsley, who performed the first operation, in the *Therapeutic Gazette*, March 15, 1911. Patient was a negro, aged thirty-seven years. When first seen presented a large bulging mass over the second and third right interspace, which showed decided expansile pulsation and a loud continuous bruit. The *x*-ray examination revealed what was apparently a sacculated aneurysm of the ascending portion of the arch of the aorta. There was continuous pain in the right upper chest, occasionally shooting into the right arm and the right side of the neck. He was first wired October 24, 1909 (eighteen feet of wire being used), the patient remarking that the pain was less shortly after the current began to pass. During the seven weeks he remained in the hospital at this time he complained of no discomfort or pain except after exercise. The patient presented himself at the Jefferson Hospital, nevertheless, on the day of his discharge from the Philadelphia Hospital, as he found himself unable to work and wished to escape the cold weather. We were also glad to have him for demonstration purposes. He had some cough at this time, and at the costal sternal junction of the third rib a pulsating mass was present, which was noted as softer in its upper portion, presenting also a blowing, systolic murmur. There were also some musical rales heard over both lungs. The systolic blood pressure in his left arm was 115 as against 125 in his right. These

symptoms became temporarily better for a few days. A week later, however, the mass was found to be growing larger and seemed to be pointing at the place where the wire had been introduced. The pain recurred three weeks after admission, and seven weeks after admission the mass was found to have greatly increased in size; the pain in the right arm had returned. Three months after the first operation I operated, choosing the point at which the tissues seemed thinnest and most yielding, and at which expansible pulsation was most marked. This is the only case in which during the operation the patient complained of discomfort and pain while the current was passing. Following the operation the patient was more comfortable for about ten days and then the tumor began to enlarge rapidly and the pain increased. The measurements of the mass at this time were 10.5 cm. horizontally and 8.5 cm. vertically. Death occurred by rupture of the sac externally three weeks and four days after the second operation. This case is of interest because the autopsy showed a burst in a false aneurysm, which had resulted from the escape of blood from a fusiform aneurysm of the aorta. Notwithstanding the fact that the history of the case as here detailed would seem to indicate that the patient was not greatly relieved of pain, it is nevertheless a fact that he repeatedly declared that he would gladly have the operation repeated because of the relief from the pain and distress which temporarily resulted therefrom. Possibly some of his pain was due to a second aneurysm about eight inches below the first, which was discovered at autopsy.

In a case previously reported the growth involved the ascending aorta and innominate artery (Fig. 8). No picture of the patient has heretofore been published.

Conclusions. 1. The fact that no accident has occurred in any of the twenty-two operations I have performed leads me to believe that this very radical operation is not a dangerous one, the more so as I have no knowledge of any accident having taken place when the operation was properly performed by others. In one unreported case an attempt to wire a fusiform aneurysm of the carotid artery resulted almost immediately in hemiplegia, and in another unreported case the mistake of using the negative pole in the growth loosened the clot already present and caused multiple emboli.

2. The operation is the only one that offers material hope of prolonging life, this having been prolonged in one case at least five years.

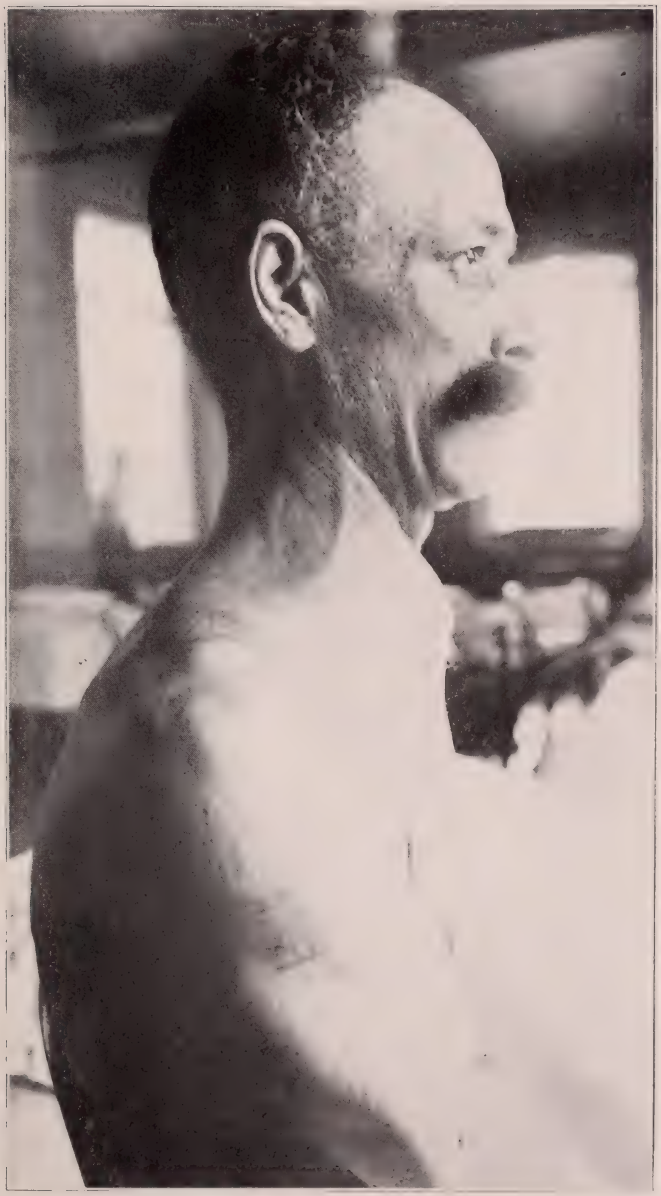

FIG. 8.—Aneurysm of the ascending aorta and innominate artery treated by wiring and electrolysis. At autopsy, four months later, no trace of the wire could be found. The patient must have pulled it out. (This case has been reported in the Therapeutic Gazette for July, 1905, but without this picture.)

3. It is justified by the prompt relief of pain which nearly always ensues and lasts, unless the growth, extending in another direction, creates a new source of pain.

4. The diagnosis of saccular aneurysm should always be confirmed by *x*-ray examination, as this operation is contraindicated in fusiform aneurysm, for obvious reasons.

5. Great care that properly prepared wire is employed is absolutely necessary.

DISCUSSION

Dr. Alfred Stengel: I have certain views regarding the wiring of aneurysm of the arch of the aorta which do not altogether agree with those of Dr. Hare, but which I should like to offer. Everyone must agree with him that there are some cases of aneurysm in which the pain and general distress are so great that any plan of treatment, even a desperate one, is at times justifiable. If there is a remote chance of some relief, I feel that this remark applies so far as the operation of wiring is concerned. But in cases of aneurysm of the arch, occasional successes should not be made the basis of a general adoption of this treatment even when there is a lack of urgent distress or pain or when the actual anatomical conditions cannot be determined as favoring the success of the operation. The only class of cases in which I feel that the conditions are such as to be entirely favorable is the one which has been referred to by Dr. Hare, namely, the projecting or bulging aneurysm. It is frequently thought that these represent merely bulging of the wall of the aneurysm and separation or erosion of the ribs. On the contrary most of them are cases of false saccular aneurysm outside the chest wall, connected with the true aneurysm inside by a very narrow passageway between the ribs or even through the bone. The introduction of wire into the external sac could, therefore, very easily prevent rupture, and as Dr. Hare has pointed out, very excellent results have been obtained in some such cases. When we come to deal with aneurysms not projecting or bulging, that an aneurysm wholly within the chest itself, it is difficult or impossible to determine to what extent they are saccular and to what extent merely ectatic or fusiform. If it were possible by physical diagnosis, by *x*-ray examination, or otherwise to make out that a given aneurysm is purely saccular, the operation by wiring could be undertaken with hopeful feelings of success and without any thought of unfavorable results. Knowing, however, that in some cases the aneurysm is purely

ectatic or is connected with the arch of the aorta by such a wide opening that the sac is practically all one cavity, the introduction of wire could easily block the direct stream coming from the heart, and it is possible also that the wire may be passed down into the ventricle or into the aorta below the sac. I present here a specimen which shows one of these results. A patient who was under my care requested wiring and this was done by one of my surgical colleagues. It is true that the wire was not most satisfactory, not coiling sufficiently when introduced, but it was the only sort that could be obtained at the time. It will be seen in inspecting the specimen that the wire projected downward into the ventricle and one strand is seen attached by a fibrinous clot to one of the tendons of the mitral valve. There was immediate relief after the operation, but within twelve hours after the completion of it a pulsation was noted in the region of the axilla. This increased for a time and then became less marked. Inspection of the specimen will show that this was a false aneurysm outside due to a rupture of the sac caused by the clotting anteriorly when the wiring was performed. This specimen, therefore, represents two of the dangers—that of having wire enter portions of the circulatory system which it is not desired to enter and having a sudden spread of the aneurysm in another direction when the clotting has prevented its extension in the direction in which the wiring is practised. This last result occurred in one other case under my observation. In this instance after wiring an aneurysm of the innominate artery the sac suddenly extended posteriorly with very serious evidences of pressure.

My feeling regarding the wiring of aneurysms is, therefore, one of doubt and hesitancy. The occasional successes are perhaps no more frequent or convincing than are the cases of spontaneous arrest by clotting, as happened in the striking instance reported by Dr. Hamill, in which a woman with a large aneurysm of the arch of the aorta lived thirteen years, and during a large part of this time continued at her occupation as a washerwoman and was able to survive a grave operation for a large ovarian tumor. I have myself seen several equally marked cases of cure by spontaneous clotting, though the length of life after the beginning of the aneurysm was not so certain as in Hamill's case.

Unless, then, the extreme degree of the pain or oppression forces one to adopt this operation, my judgment is that the dangers of the operation and the uncertainty of its usefulness should debar it.

Dr. Augustus A. Eshner: While, as a rule, life is not prolonged more than a short time, perhaps for weeks or months in these cases, and while this period has been extended over two or three years in a few instances (Stewart, three and a half years), and even up to five years in one instance (Hunner), I am informed by Rosenstein, of San Francisco, that in his case life was prolonged for more than twenty years by such an operation.

Dr. Hare, closing: In my paper, which I did not read, I cover some of the points which Dr. Stengel has brought forward. As I have said before, the general condition of the arteries is to be thought of. With widespread arterial disease you can expect only temporary improvement. The condition may be much like mending a rotten hose. If the aneurysm is due to trauma and widespread disease is not present there is much chance of the operation doing good. With deflection of the blood stream posteriorly the wall may give way in that direction and produce death. In 1 case wiring was done by my assistant, and I did a second wiring. There was present a false aneurysm connected with the true aneurysm underneath, but the patient was wonderfully relieved of pain. Death occurred later, however, from rupture. Autopsy showed another aneurysm eight inches below the first near the diaphragm, which was never suspected during life.

EXPERIMENTAL STUDIES ON THE ADMINISTRATION OF SALVARSAN BY MOUTH TO ANIMALS AND MAN[1]

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The experimental studies here described were undertaken with the view of determining whether salvarsan could be administered by mouth without producing poisonous effects, and whether, when so given, it exerted a therapeutic influence on the manifestations of syphilis. Certain studies incidental to the oral administration of salvarsan have also been made which may prove not to be devoid of interest.

ANIMAL EXPERIMENTATION

Before any attempt was made to administer salvarsan to patients by mouth the drug was given to experimental animals to determine whether or not it would produce deleterious effects. It was also necessary to prove that the medicament thus given was absorbed.

In administering the drug by mouth to animals we sought to determine four points: (1) The primary and secondary toxic effects, if any, of a single large dose and of several small doses

[1] Read March 6, 1912.

of the medicament; (2) the presence or absence of arsenic in the bile and urine and in the stomach and intestinal contents; (3) the influence of salvarsan upon the bacterial flora of the intestinal tract, especially upon that of the colon; (4) the effect of the drug upon the parenchymatous organs, especially those concerned in elimination.

TOXIC EFFECTS

To determine the primary effects of salvarsan given by mouth, the drug was administered in various forms to a series of cats, rabbits, and dogs. It was given in pills, in capsules, and dissolved in milk and water.

EXPERIMENT 1.—A cat, weighing 2650 gm., was given 0.01 gm. of salvarsan dissolved in 10 c.c. of milk and water. No other food was given for twenty-four hours. There was neither vomiting nor diarrhea. The cat was killed at the end of twenty-four hours for further study.

EXPERIMENT 2.—A cat, weighing 3250 gm., was given 0.01 gm. of salvarsan dissolved in 10 c.c. of warm milk and water. No other food was given. The animal showed no toxic effects and was killed and autopsied forty-eight hours later.

EXPERIMENT 3.—A cat, weighing 2550 gm., received 0.05 gm. of salvarsan dissolved in 10 c.c. of milk and water. The animal showed no toxic effects.

EXPERIMENT 4.—A cat, weighing 850 gm., received 0.05 gm. of salvarsan in a capsule. The animal was autopsied twenty-four hours later, but showed no evidence of toxic action.

EXPERIMENT 5.—A cat, weighing 2700 gm., received 0.05 gm. of salvarsan in a capsule. The animal showed no evidence of toxic effects and was autopsied twenty-four hours later. The findings were negative.

EXPERIMENT 6.—A rabbit, weighing 2080 gm., received 0.05 gm. of salvarsan dissolved in 20 c.c. of milk and water. The animal showed no evidence of toxic effects and was killed eighteen hours later and autopsied.

EXPERIMENT 7.—A rabbit, weighing 2000 gm., received 0.05 gm. of salvarsan dissolved in 12 c.c. of milk and water. The animal showed no evidence of toxic effects and was autopsied at the end of forty-eight hours.

EXPERIMENT 8.—A rabbit, weighing 1380 gm., received 0.05 gm. of salvarsan dissolved in 12 c.c. of milk and water. The animal showed no toxic effects, and was autopsied seventy-two hours later.

EXPERIMENT 9.—A rabbit, weighing 1450 gm., received 0.05 gm. of salvarsan in a capsule. The animal showed no toxic effects, and was autopsied twenty-four hours later, with negative findings.

EXPERIMENT 10.—A rabbit, weighing 1400 gm., received 0.05 gm. of salvarsan in a capsule. The animal showed no toxic effects and was autopsied forty-eight hours later. The findings were negative.

EXPERIMENT 11.—A rabbit, weighing 1350 gm., received 0.008 gm. of salvarsan in a capsule. The animal was autopsied ninety-six hours later. It had shown no toxic effects, and the autopsy findings were also negative.

EXPERIMENT 12.—A rabbit, weighing 1400 gm., received 0.008 gm. of salvarsan in a capsule. The animal was autopsied ninety-six hours later. It had shown no toxic effects, and the autopsy findings were also negative.

EXPERIMENT 13.—A cat, weighing 2550 gm., received 0.05 gm. of salvarsan dissolved in 10 c.c. of milk. The animal showed no primary toxic effects and at the present time is apparently in a normal condition.

EXPERIMENT 14.—A cat, weighing 3550 gm., received 0.02 gm. of salvarsan dissolved in 20 c.c. of warm milk. The animal did not show any primary toxic effects and at the present time apparently is in good health.

EXPERIMENT 15.—A cat, weighing 2850 gm., received 0.04 gm. of salvarsan dissolved in 40 c.c. of warm milk. The animal showed no toxic effects and was autopsied forty-eight hours later.

EXPERIMENT 16.—A dog, weighing 5700 gm., received 0.025 gm. of salvarsan in the form of a pill. No toxic effects were seen, and the dose was repeated in twenty-four hours without producing any deleterious effects.

EXPERIMENT 17.—A dog, weighing 7000 gm., received a pill containing 0.025 gm. of salvarsan and this was followed by 10 c.c. of milk. The dose was repeated at the end of twenty-four and forty-eight hours without producing any appreciable toxic effects.

EXPERIMENT 18.—A dog, weighing 7200 gm., received in the form of a pill 0.01 gm. of salvarsan every day for twenty-two days, followed by 0.05 gm. in a capsule each day for the next seven days. The animal did not lose weight, its appetite and spirits remained perfectly normal, and at no time were toxic effects seen.

These experiments show that salvarsan is without immediate toxic effects when administered to animals by mouth in a relatively

large single dose or in multiple smaller doses. In some of these experiments six weeks have elapsed since the drug was given, and the animals have shown no late or secondary poisonous effects attributable to arsenic. The solutions were made and administered while fresh, without the addition of an alkali.

Many of the pills had been made up as long as fifteen days before administration and had become very dark in color and were quite hard. These were crushed in a mortar before administration but in no instance could harmful effects be noticed.

The capsules, which contained diatomaceous earth with the salvarsan, did not undergo any change of color although exposed to light for several weeks.

ABSORPTION AND ELIMINATION

The intravenous and intramuscular administration of salvarsan are followed by the elimination of arsenic in the urine and feces. So far there are no reliable tests for salvarsan *per se* in any of the excretions. The question of absorption of salvarsan from the intestinal tract is worthy of study. Whether or not the drug undergoes marked changes in the intestinal canal is as yet undetermined. We have examined the secretions of a number of experimental animals in order to determine whether the drug was being eliminated as arsenic. If arsenic were found, this would indicate absorption of the drug under these conditions.[1]

TECHNIQUE. All tests were qualitative. Suitable controls were used. In brief the materials (stomach contents, feces, bile, and urine) were oxidized with concentrated nitric and sulphuric acids, after which sodium and potassium nitrate were added and the whole mass was fused with more of the same salts. The resulting product was dissolved in concentrated sulphuric acid over a water bath until all nitric acid had disappeared. The March test was then employed.

[1] We are indebted to Professor Alonzo E. Taylor and Dr. Ringer, of the Department of Physiological Chemistry of the University of Pennsylvania, for assistance and suggestions in conducting these tests.

The results are given in the following table:

TABLE I.—Tests for Arsenic

No. of experiment.	Animal.	Dose in gram.	Time of removal after administration.	Product.	Arsenic present.
3	Cat	0.05	24 hours	Stomach	Well-defined trace
				Feces	Well-defined trace
				Bile	Trace
				Urine	Trace
6	Rabbit	0.05	24 hours	Stomach	Well-defined trace
				Feces	Well-defined trace
				Bile	Trace
				Urine	Trace
7	Rabbit	0.05	48 hours	Stomach	No trace
				Feces	Trace
				Urine	Very slight trace
8	Rabbit	0.05	72 hours	Stomach	No trace
				Feces	No trace
				Bile	No trace
				Urine	No trace
18	Dog	0.01	After 3 doses	Feces	Well-defined trace

These results show that within twenty-four hours after the oral administration of salvarsan, arsenic may be found in the bile and urine. Its presence in the stomach contents and feces may be indicative of failure of absorption. At the end of forty-eight hours the quantities of arsenic found were very minute, and after seventy-two hours none was found in the stomach, feces, bile, or urine. It is probable that only a portion of the salvarsan was absorbed from the intestinal canal, and that this was quickly eliminated largely in the form of arsenic.

INFLUENCE ON INTESTINAL BACTERIA

As a matter of additional interest, a quantitative examination of the bacteria of the gastro-intestinal canal was made in a few of the experimental animals, since arsenic and possibly some of the unchanged drug had been found in the feces twenty-four hours after the oral administration of salvarsan.

TECHNIQUE. For quantitative work with intestinal bacteria, the question of a satisfactory technique was most important. The animals were usually kept from all food for twenty-four hours

before and after the administration of salvarsan. As a result, the stomach was usually empty at autopsy. Cultures were made of the stomach, ileum (close to the ileocecal valve), and the upper portion of the rectum. After asepticizing the gut with a hot iron, the wall was cut through and cultures were secured by rubbing the wall or contents with a sterilized platinum loop which measured 4 mm. in diameter. The loop was then thoroughly shaken in 10 c.c. of sterilized salt solution. This solution was thoroughly emulsified and 0.5 c.c., 1 c.c., and 2 c.c. were plated with 10 c.c. of plain neutral agar. The plates were incubated at 37° C. for twenty-four hours and then kept at room temperature until the end of the forty-eighth hour, when counts were made. The control animals were living under exactly the same conditions and receiving the same diet. From them likewise food was kept for forty-eight hours preceding the autopsy and the taking of the cultures.

TABLE II.—The Results of Quantitative Bacteriological Examination of the Intestinal Tract of Animals Receiving Salvarsan by Mouth

No. of experiment.	Animal.	Dose in gram.	No. of hours separating the administration of the drug and the taking of cultures.	Organ.	No. of colonies of bacteria per c.c. of emulsion.
1	Cat	0.01	24	Stomach	10
				Ileum	450
				Colon	14,000
3	Cat	0.05	24	Stomach	None
				Ileum	270
				Colon	10,500
15	Cat	0.04	48	Stomach	18
				Ileum	1,200
				Colon	17,000

TABLE III.—Table of Control Bacteriological Examinations

No. of control.	Animal.	Organ.	No. of colonies of bacteria per c.c. of emulsion.
1	Cat	Stomach	24
		Ileum	11,000
		Colon	19,000
2	Cat	Stomach	19
		Ileum	12,200
		Colon	20,000
3	Cat	Stomach	50
		Ileum	17,000
		Colon	19,500

While the number of cats from which cultures were made was comparatively small, the results were fairly constant. The most marked influence was seen in the lower end of the ileum, where, within twenty-four hours after the administration of salvarsan, the total number of bacteria was found much decreased. In the colon, the decrease in the number of bacteria was about 30 per cent. At the end of forty-eight hours, however, the differences were less marked.

EFFECTS OF SALVARSAN ON PARENCHYMATOUS ORGANS, ESPECIALLY THOSE OF ELIMINATION

URINARY EXAMINATIONS. The urine of the six animals that received salvarsan by mouth, Nos. 4, 5, 9, 10, 11, and 12, and of four others, Nos. 19, 20, 21, and 22, that received the drug intravenously, was examined, but in no instance was the presence of albumin or casts demonstrated, the findings being, in fact, entirely similar to those in the normal controls. In all the animals mentioned, the special test for salvarsan in the urine advocated by Abelin was tried. In these tests we secured a clear dark brownish discoloration in all specimens of urine from the experimental and control animals, but in no instance did we find the red color described as a positive reaction in this test.

AUTOPSY PROTOCOLS. Twelve of the eighteen experimental animals were autopsied at intervals varying from eighteen to ninety-six hours after the administration of salvarsan. In none of these animals were there any gross evidences of deleterious effects. The heart, lungs, liver, spleen, kidneys, and the mucosa of the stomach and of the entire intestinal tract were found to be normal. The liver and kidneys of many of the animals were examined in the fresh condition for albuminous degeneration of the cells, but the findings did not differ from those of the control animals. The liver of one cat (experiment 4) had the gross appearance of fatty changes, but a control animal showed an exactly similar condition.

Histological Examination. Sections of the heart, lungs, liver, spleen, kidneys, stomach, and ileum from experimental animals 1, 2, 4, 5, 9, 10, 11, and 12 were fixed, stained, and examined histologically for evidence of vascular disturbances and degenerative changes. After examining the sections from animals 1, 2, 4, and 5, which were cats, we at first thought that, in addition to the fatty infiltration found normally in a large proportion of cats, important albuminous and fatty degenerative changes had been produced. But after studying the organs of three control cats living under the same conditions, it was found that no degenerative or vascular changes could be ascribed to the influence of the salvarsan.

In order to study this question more thoroughly, salvarsan was administered intravenously in alkaline solution to the following four rabbits:

Experiment 19.—A rabbit, weighing 1500 gm., received intravenously 0.006 gm. of salvarsan dissolved in 16.8 c.c. of water. The animal was autopsied forty-eight hours later.

Experiment 20.—A rabbit, weighing 2500 gm., received intravenously 0.01 gm. of salvarsan dissolved in 28 c.c. of water. The animal was autopsied ninety-six hours later.

Experiment 21.—A rabbit, weighing 1400 gm., received intravenously 0.0056 gm. of salvarsan dissolved in 15.6 c.c. of water. It was autopsied eighteen hours later.

Experiment 22.—A rabbit, weighing 1000 gm., received 0.004 gm. of salvarsan dissolved in 11.2 c.c. of water, and was autopsied twenty-four hours later.

According to body weight, the doses administered were about equal to those administered intravenously to man. None of these animals showed any toxic effects whatsoever, the urine examinations were negative, and the gross autopsy findings were entirely negative. Sections of the heart, lungs, liver, kidneys, spleen, stomach, and ileum of these four rabbits and of rabbits 9, 10, 11, and 12, were examined histologically and contrasted with preparations of the organs from our normal rabbits. While the sections from the hearts showed in some instances a loss of muscle

striations and fragmentation of the muscle cells with slight fatty changes, entirely analogous changes were found in the controls. The lungs showed no changes. The stomach and ileum appeared entirely normal, there being no evidences of vascular changes or degeneration of cells. Sections of the liver showed in many instances marked granularity of the cells, with perivascular interstitial connective-tissue proliferation, but in the normal controls entirely analogous conditions were found. The kidneys showed some cloudy swelling of cells in a few of the sections, but no vascular changes. The controls were similar. The spleen showed in a few instances a large number of eosinophile cells and occasionally apparent enlargement of the Malpighian bodies. There was no congestion or endothelial cell proliferation. The normal controls were similar in all particulars.

As a result of these studies, we must conclude that salvarsan in the doses in which we administered it does not produce appreciable degenerative changes in the principal organs within ninety-six hours following its administration.

ORAL ADMINISTRATION OF SALVARSAN TO MAN

As it had been demonstrated that salvarsan could be given by mouth to cats, dogs, and rabbits in relatively large doses without producing toxic effects, or, indeed, untoward symptoms of any kind, we proceeded to administer it to human subjects.

A decigram of salvarsan dissolved in hot milk was first given to three men suffering from secondary and tertiary syphilis. In two of these patients the drug was well borne, no symptoms of gastro-intestinal irritation and no other disturbing symptoms developing.

Case I.—In a man, aged thirty years, vomiting, followed by diarrhea, occurred two hours after the ingestion of the salvarsan, but the patient had no pain and was not otherwise disturbed.

On the following day a second dose of the same amount was administered to this patient without producing vomiting or diarrhea. Four

days later, 0.2 gm. of salvarsan dissolved in hot water to which a little peppermint water was added, was given. This was followed by some vomiting and diarrhea, but the patient had no pain and did not feel ill in any way. On the two succeeding days, he received 0.2 gm. and 0.15 gm. respectively without vomiting or diarrhea.

This patient had an extensive small papular syphilide (follicular syphilide), for which he had a short time before received nine mercurial inunctions, with but slight influence on the eruption. A mucous patch on the lip healed promptly after the salvarsan was given and the eruption was perceptibly improved.

CASE II.—A man, aged twenty years. He had a chancre and a macular syphilide with beginning papular lesions.

On January 21, 1912, the patient was given 0.1 gm. of salvarsan in 90 c.c. of milk. On January 20 the dose was repeated. On January 24, 0.2 gm. was given in water with pepermint. On January 25, 0.2 gram was again administered.

All of these doses were well borne, the patient experiencing no unpleasant symptoms whatsoever. The treatment was discontinued owing to a misunderstanding of orders.

During the administration of the drug, the macular eruption disappeared, but a large flat papular syphilide continued to make its appearance.

CASE III.—The patient was a man, aged thirty-eight years. He has been a hard drinker. Twelve years ago he had a chancre and secondaries, and then had received mercurial inunctions, but in the last twelve years he has received no treatment. The patient has a large ulcerating gumma below the knee which is deeply excavated and discharges profusely (Fig. 1). The gumma has existed since September, 1911.

Treatment. On January 19, 1912, and again on January 20, 0.1 gm. of salvarsan was administered in milk. Healthy granulations were observed on January 20. On January 25, 0.2 gm. of salvarsan was administered in water, and on this date the first photograph (Fig. 1) was taken. On January 26, 0.2 gm. of salvarsan was given in water. On January 27 the gumma was healing quickly, and granulations were appearing rapidly. On January 27, 28, and 29 a capsule containing 0.1 gm. of salvarsan and 0.2 gm. of diatomaceous earth was given.

On February 2 the ulcer was about half its former size. The granulations were on a level with the skin. The discharge was very scanty, and cicatrization from the border was advancing. The local treatment consisted merely of the employment of a wet boric acid dressing. The patient stated that he felt well and that his appetite was most keen.

On February 2, the full dose of 0.6 gm. of salvarsan in 120 c.c. of water flavored with peppermint was administered. No unpleasant symptoms followed. The ulcer was healing rapidly.

FIG. 1. Case 3.—First photograph, taken January 25, 1912. Gummatous ulcer developing twelve years after infection. No treatment was given after the initial inunctions. This patient received 2.4 grams of salvarsan by mouth within a period of four weeks.

On February 10 another full dose of 0.6 gm. of salvarsan was given. No unpleasant symptoms followed.

The second photograph (Fig. 2) was taken on March 1, just five weeks

after the first. It shows the ulceration to be practically healed, although epidermization is not yet complete.

FIG. 2. Case 3.—Second photograph, taken March 1, 1912.

CASE IV.—A young woman, with a recurrent papulosquamous syphilide of the wrists and palms, received 0.1 gm. of salvarsan in a capsule, once a day for ten days. The drug was well borne, but as the improvement was but slight, intravenous administration was resorted to.

Case V.—A young negro, with a beginning follicular syphilide on the trunk and an annulopapular syphilide of the penis and scrotum, received the full dose of 0.6 gm. of salvarsan in 200 c.c. of water. About three-quarters of an hour later he vomited, and after this had seven loose bowel movements.

It was subsequently ascertained that prior to the ingestion of the drug he had had no food for eighteen hours. Three days later the follicular syphilide had almost disappeared, but the patches on the penis and scrotum were but slightly influenced.

A week after the first administration, a second dose of 0.6 gm. of salvarsan in 200 c.c. of water was given. This was followed by slight nausea, but no vomiting. The patient had four loose bowel movements, but no soreness or pain.

After the second dose of salvarsan, the genital eruption improved but did not disappear. Other treatment was therefore substituted.

Case VI.—The patient was a woman, aged forty years, who had on her back a palm-sized tubercular syphilide that had come on three years after infection. She received four capsules of 0.1 gm. of salvarsan on successive days, and three days later took 0.6 gm. dissolved in 200 c.c. of water. The patient had two loose bowel movements, but no vomiting. There was practically no improvement in the eruption.

Case VII.—A mulatto girl, aged nineteen years, with an extensive pustular syphilide of the varicelliform type, was given 0.6 gm. of salvarsan in 200 c.c. of water. Three hours later she vomited twice, and had some cramps, but no diarrhea. Three days later, 0.6 gm. of salvarsan was again administered. Some hours later the patient had nausea and slight vomiting.

The influence of the salvarsan on the eruption in this patient was extremely slight.

Urinary Examinations. Twenty-four hours after the full doses of salvarsan had been administered by mouth, the urine of the patients treated by mouth was subjected to careful chemical and microscopic examination. Albumin was tested by the nitric acid, heat, and potassium ferrocyanide tests and was absent in all the examinations except one in which a trace was disclosed by the potassium ferrocyanide reaction only. In this specimen of urine a single hyaline cast was found. With this exception the microscopic examinations were negative.

SUMMARY

Salvarsan in capsule and in solution in doses varying from 0.1 to 0.6 gram was administered by mouth to seven patients suffering from syphilis. Five patients were given full doses of 0.6 gram. One patient (Case III) received in the aggregate within four weeks 2.4 grams without loss of weight or any disturbing effects. An ulcerating gumma of the knee was practically healed after a period of five weeks. This was the only patient of the seven, however, in whom notable improvement in the cutaneous manifestations followed the administration of the drug. This ulceration would probably have healed as soon, or possibly sooner, under the influence of mercury and the iodides.

From the above observations it would appear that salvarsan administered by mouth up to the full dose of 0.6 gram has a perceptible though feeble therapeutic effect on cutaneous syphilitic manifestations. We should naturally expect the influence of any drug to be more evident in the case of a gumma, in which spirochetes are only sparsely present, than in the secondary syphilides in which the spirochetes are present in enormous numbers. Cutaneous gummata are readily healed at times by very small doses of mercury or iodides.

It is interesting to note that the administration of the full dose of the drug by mouth does not appear to give rise to symptoms of systemic arsenical poisoning, unless the vomiting and diarrhea be regarded as such. After some of the full dose administrations there was neither vomiting nor diarrhea. As salvarsan contains 34 per cent. of arsenic, these patients received the equivalent of five grains of arsenic. In Case III, in the course of four weeks the patient took by mouth the equivalent of over twelve grains of arsenic, without vomiting or having diarrhea, and with no loss of weight or disturbance of health. The urine of this patient was entirely normal. It would seem, therefore, that salvarsan is not converted in the gastro-intestinal tract into an organotropic arsenical compound, and it is possible that it may remain practically unchanged.

CONCLUSIONS

1. Salvarsan can be administered in pills, in capsules, and in solution to lower animals in dosage of 0.02 to 0.03 gm. per kilo of body weight, without producing toxic symptoms. A dog received 0.57 gm. of salvarsan in pill and capsule form in the course of twenty-nine days without any disturbing effects.

2. After the oral administration of salvarsan, arsenic is found in the bile and urine at the end of twenty-four hours, but it disappears by the end of seventy-two hours.

3. Twenty-four hours after the oral administration of salvarsan to cats, the number of bacteria in the intestinal tract appears to be reduced, the reduction being most striking in the lower end of the ileum.

4. Salvarsan administered by mouth and likewise intravenously to rabbits in doses approximating those employed in human subjects, does not produce, at least within ninety-six hours following its administration, any appreciable microcopic changes in the important viscera.

5. With the exception of a little vomiting and diarrhea in some cases, salvarsan can be given by mouth up to 0.6 gm. to human subjects without producing toxic symptoms. Thus administered, the drug exerts a therapeutic influence, but this influence is too feeble to warrant its use by this route.

THE PROGNOSTIC SIGNIFICANCE OF THE ATROPINE REACTION IN CARDIAC DISEASE[1]

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My interest in the influence of the vagus on the rate of the heart beat was awakened last year while working in Dr. James Mackenzie's wards in the Mt. Vernon Hospital, London. For the many courtesies shown me by Dr. Mackenzie, Prof. Cushny, Dr. Marris, and other coworkers in the clinic I am deeply grateful. The work on which this paper is based was done largely during my term of service last year in the Presbyterian Hospital, Philadelphia. I appreciate the kindness of my colleagues of the medical staff who placed material at my disposal. It is regrettable that suitable material and the necessary leisure to use it so often fail to come together in point of time.

The recapitulation of a few facts concerning the normal physiological influence of the vagus on the rate of the heart beat may not be out of place. Stimulation of one or both vagi by a rapidly interrupted induction current slows or stops the heart. Such vagal stimulation lessens the conductivity, but does not destroy the irritability of the heart, since direct strong mechanical stimuli may provoke a beat even during cardiac standstill due to vagal stimulation. Conversely, section of one vagus produces some acceleration of the pulse, section of both vagi causes greater acceleration. These phenomena are constant in the vertebrates and prove that the vagi are the normal paths by which travel continually restraining impulses from the inhibitory

[1] Read March 6, 1912.

centres in the medulla to the heart. Physiologists have also found it a constant experience that atropine produces a decided increase in the cardiac rate by paralysis of the vagi. Clinicians find that a hypodermic injection of grain $\frac{1}{50}$ to grain $\frac{1}{25}$ of atropine produces the same effect. Physiologists have further shown that atropine acts on the vagi, and not on the muscular substance of the heart, by the observation that in early embryos, so young that no nerves have yet grown to the heart, the drug has little or no effect.[1]

After making dozens of injections of atropine, grain $\frac{1}{25}$ in robust males and grain $\frac{1}{50}$ in females and less robust males, the writer has yet to see any untoward effect, or hear any decided complaint. The patient should be told that the throat will become dry, and ability to read may be lost for part of the day. Almost always the patient is ready for the next meal, rarely complains of insomnia, and the following day reads as usual. The same has been true in those cases of rebellious hemoptysis where the same doses of atropine have been used.

Professor Cushny suggested the releasing of the vagus by means of hypodermic doses of atropine, so-called atropine reaction, in the study of digitalis action. Silberberg[2] made use of it in studying the action of digitalis in cardiac disease, especially auricular fibrillation. His paper will be referred to later. The method of taking the reaction is thus: The recumbent patient is made as comfortable as possible, the arm supported on a pillow or folded sheet, the ink polygraph applied to the right radial, the engine started at slow speed and the pulse rate taken graphically for about ten minutes, so as to get an average pulse rate before atropine. Throughout the minutes are marked off by touching the time marker every minute by the watch. This makes a slight break in the line, which simplifies the counting later. The hypodermic of atropine is now given in the other arm, while the engine is still running, and the pulse rate taken continually for another

[1] Halliburton, Handbook of Physiology, p. 256.
[2] Proc. Royal Soc. Med., iv, 192 (1910–1911).

fifty to sixty minutes. The influence of the drug, ordinarily dry throat, may appear as early as twelve to fifteen minutes, but on

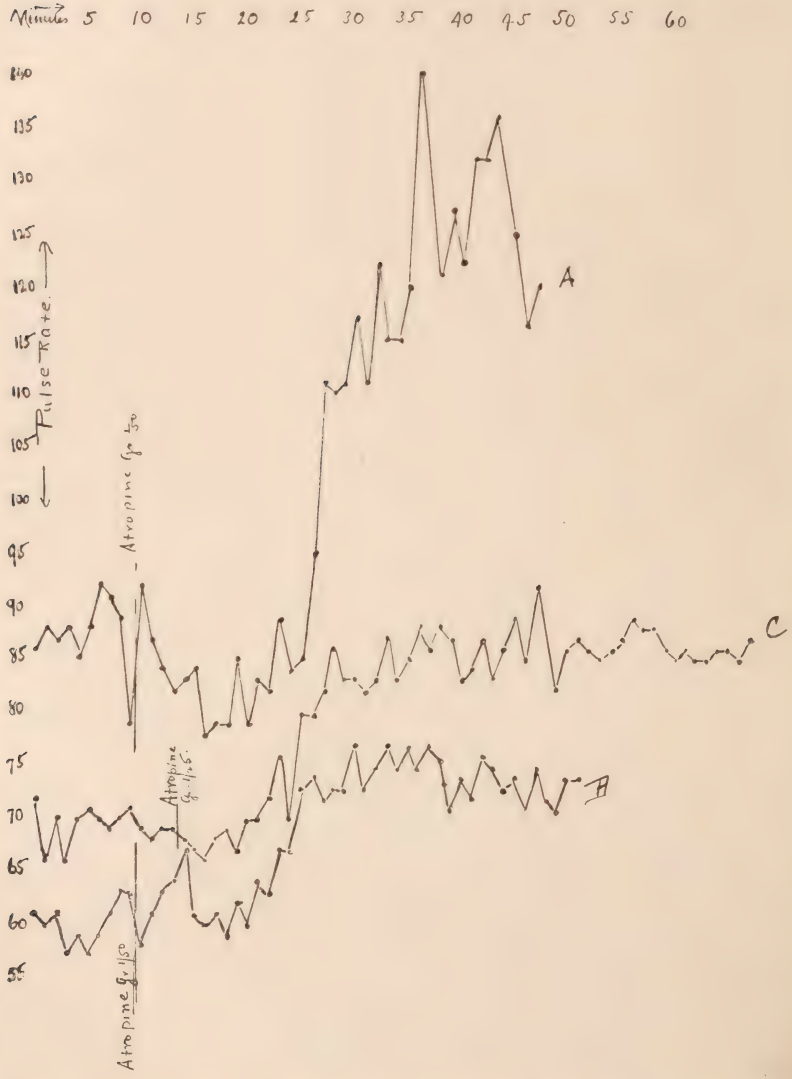

FIG. 1.—Curve *A* represents the influence of atropine, grain $\frac{1}{30}$, in Case I, Table II, before digitalis. Curve *B* represents the influence of atropine, grain $\frac{1}{30}$, in Case I, Table II, on full digitalis. Curve *C* represents the influence of atropine, grain $\frac{1}{25}$, in Case No. 14, Table I.

the average in twenty minutes. The maximum is usually reached in from twenty to thirty minutes from the time of injection, and usually there is no further rise after this or at least after the hour. Just how long the influence may last I have not had time to determine, but in a few cases, when taken at three to five minute intervals, during the second hour there has been no further rise. Counting the pulse rate from the long rolls of paper and setting them down in order in a column shows at a glance when the maximum is reached. The graphic representation by means of loci curves (Fig. 1) shows the contrasts better. The pulse rate before atropine is the average for the ten minutes before the hypodermic. The reaction or the pulse rate after the atropine is determined in this paper by taking the average for ten minutes when the pulse rate is at its maximum after the injection.

The writer started out with two ideas: One was to determine the atropine reaction in hearts as normal as the material available furnished. Of course, the medical wards of a hospital are not ideal places to look for absolutely normal hearts, yet many are approximately so. The individual outside with a perfect cardiovascular apparatus is not keen to lend himself to such determinations, because it may mean loss of time if the atropine happens to paralyze accommodation. Having obtained four reactions in persons with approximately normal hearts, there was no difficulty in obtaining plenty of contrasting material, as will be shown by Table I. The other idea was to use the reaction in the study and treatment of patients with cardiac disease. Unfortunately too many of the available cases were of the regular pulse type. However, several studies will be embodied in Table II.

Table I.—The patients in Table I, except the last four, had at least no gross lesions of the heart. Nos. 1 and 4 were apparently entirely normal, and Nos. 2 and 3 were so far from their original attacks as to be practically so. They were having extension or superheated air for the old lesions in the joints. Taking these 4, though inadequate, I know, for a working basis, we may look upon the normal reaction as from 30 to 40. The actual average of the 4 is 34. The next ten observations were made in

patients who had grossly normal hearts, however these hearts had all been subjected to a chemical poison (No. 5), or the toxins and fever of enteric (Nos. 6 to 10). As the pulse rate, the vagal influence being withdrawn, depends upon the inherent activity of the rhythm giving portion of the heart, it was expected there might be a difference in reaction, and, broadly speaking, this was found true. The exhausted cardiac tissue showed a lessened rather than an increased irritability. Observation 5a was clear when the fault in technique was discovered. In 5b the small reaction is probably due rather to the toxic material in the circulation, due to the acute kidney irritation, rather than from any influence of the possibly absorbed bichloride of mercury itself on the heart muscle. This view is supported by the normal reaction obtained in observation 5c, the kidneys having cleared up in the two weeks intervening. All the enteric cases (Nos. 6 to 10) give a reaction that is less than normal. In cases Nos. 6 and 7, though a week intervened between the two reactions in each case (5a and 5b, and 6a and 6b), the second reaction was less than the first in each case (6a, 24; 6b, 18; 7a, 24; 7b, 17). One would have expected that after a week, the heart having recovered somewhat, the reaction would have been greater rather than less. The same thing is evident in taking the whole group. The longer a patient has had a normal temperature the higher the reaction does not follow. For example, Case 10, with temperature normal thirty days, gives a reaction of 12, while 6a and 7a, with normal temperature for seven and nine days respectfully, give each a reaction of 24. The small reaction of these enteric cases reminds one of the similar reaction in the senile cardiosclerosis cases. Here the morbid processes leading to slight response are probably but temporary, while the degenerative process in the cardiosclerosis cases are permanent.

TABLE I

No.	Sex.	Age.	Diagnosis.	Blood pressure	Date.	Pulse rate before atropine.	Pulse rate after atropine.	Full vagal effect.	Dose of atropine.	Throat dry. Minutes.	Remarks.
1	F.	18	Hysteria	116—80	October 10	102	132	30	1/25	20	Patient well.
2	F.	21	Old gonorrheal arthritis (hand)	120—60	September 21	95	121	26	1/50	23	No fever for one month.
3	M.	25	Old gonorrheal arthritis (knee)	111—65	October 20	71	111	40	1/25	30	Acute attack months before.
4	F.	48	Gastroptosis	145—75	November 20	89	129	40	1/50	18	Patient admitted for operation.
5a	M.	24	Acute corrosive sublimate poisoning	130—75	September 27	73	68	..	1/25	..	Tablet undissolved in syringe.
5b	M.	24	Acute corrosive sublimate poisoning	..	September 28	77	93	16	1/33	20	Urine full of albumin and casts.
5c	M.	24	Acute corrosive sublimate poisoning	134—60	October 11	61	101	40	1/25	18	Urine practically normal.
6a	M.	35	Enteric fever	110—60	October 6	66	90	24	1/25	25	Seventh day of normal temperature.
6b	M.	35	Enteric fever	120—70	October 14	100	118	18	1/25	15	Fifteenth day of normal temperature. Patient up and around.
7a	F.	34	Enteric fever	100—55	November 29	104	128	24	1/50	20	Ninth day of normal temperature.
7b	F.	34	Enteric fever	..	December 6	114	131	17	1/50	17	Sixteenth day of normal temperature. Patient up and around.
8	M.	29	Enteric fever	125—60	October 17	89	109	20	1/25	24	Tenth day of normal temperature.
9	M.	21	Enteric fever	120—57	October 2	113	120	7	1/25	20	Fourteenth day of normal temperature. Patient up and around.
10	F.	18	Enteric fever	..	September 30	99	111	12	1/50	18	Thirtieth day of normal temperature. Patient up and around.
11	F.	29	Small tumor pressing on right vagus	..	June 21	66	130	64	1/50	19	Bradycardia; extrasystoles.
12	M.	45	Mitral disease; auricular fibrillation	125	August 11	101	115	14	1/25	20	
13	F.	26	Mitral disease; auricular fibrillation	..	July 28	109	143	34	1/50	20	
14	M.	60	Cardiosclerosis	170	August 19	70	87	17	1/25	16	Extrasystoles; auricular and ventricular; P-R normal.
15	F.	22	Mitral and aortic disease	108—90	September 29	68	99	31	1/50	25	Pulse always regular; venous pulse, auricular type.

TABLE II

No.	Observation.	Sex.	Age.	Diagnosis.	Blood pressure.	Date.	Pulse rate before atropine.	Pulse rate after atropine.	Full vagal effect.	Full digitalis effect.	Vagal factor in digitalis.	Tissue factor in digitalis.	Dose of atropine.	Amount of digitalis taken.	Throat dry. Minutes.	Remarks.
Ia	Before digitalis	M.	35	Mitral stenosis; auricular fibrillation	..	June 7	90	130	40	..	..	..	1/50	..	27	
Ib	Full digitalis	M.	35	Mitral stenosis; auricular fibrillation	..	June 21	61	75	..	29	14	55	1/50	3i4	18	
Ic	Before digitalis	M.	35	Mitral stenosis; auricular fibrillation	..	July 3	95	145	50	..	..	..	1/25	..	45	Two doses, grain 1.50, atropine; second half-hour after first.
Id	Full digitalis	M.	35	Mitral stenosis; auricular fibrillation	..	July 26	63	75	..	32	12	70	1/50	3i4	26	
IIa	Before digitalis	F.	20	Mitral disease; auricular fibrillation	..	June 15	112	148	36	..	..	..	1/25	..	20	
IIb	Full digitalis	F.	20	Mitral disease; auricular fibrillation	..	July 3	55	56	..	57	1	92	1/25	3i4	15	Two to one heart block.
IIIa	Before digitalis	M.	45	Mitral disease; auricular fibrillation	110—75	May 28	87	115	28	..	..	..	1/25	..	30	
IIIb	Full digitalis	M.	45	Mitral disease; auricular fibrillation	115—65	July 5	51	80	..	36	29	35	1/25	3i4	30	
IIIc	On digitalis	M.	45	Mitral disease; auricular fibrillation	85—55	June 13	85	117	..	2	32	0	1/25	3 9	24	
IVa	Before digitalis	M.	35	Cardiosclerosis; auricular fibrillation	145	July 28	60	78	18	..	..	..	1/50	..	25	
IVb	Full digitalis	M.	35	Cardiosclerosis; auricular fibrillation	..	August 31	60	60	..	0	0	18	1/50	3i2	24	
Va	Before digitalis	M.	71	Cardiosclerosis; heart block	132—65	December 17	77	96	19	..	..	..	1/50	..	20	Phases of partial block and tachycardia before digitalis.
Vb	Full digitalis	M.	71	Cardiosclerosis; heart block	172—102	January 1	42	106	..	35	64	0	1/33	..[1]	20	
Vc	On digitalis	M.	71	Cardiosclerosis; heart block	..	December 23	80	82	..	..	2	14	1/50	..[2]	20	
VIa	Before digitalis	M.	14	Heart sound; after pneumonia	95—45	December 23	73	103	30	..	..	..	1/50	..	25	Six days after crisis.
VIb	On digitalis	M.	14	Heart sound; after pneumonia	102—50	January 6	74	114	..	0	40	0	1/50	3 7	24	

[1] Infusion 15 ounces; digitalone, 3v.

[2] Infusion 7 ounces.

Case No. 2 was a patient of Dr. W. E. Robertson's, with whom I took her atropine reaction. She had a small tumor either involving or at least pressing upon the right vagus, which gave her a bradycardia. Her pulse during the day we took the reaction had been 36. The emotional disturbance of having a new procedure tried was probably responsible for the average rate of 66 before the atropine. The reaction of 64 reminds one of the response often given by cases of auricular fibrillation with a high pulse rate before atropine. In such cases the bundle is supposed to be in a highly irritable condition, and to carry too many impulses from the disorderly auricle. That her heart was irritable is shown by the greater number of extrasystoles in the tracing even during the time she was under the influence of atropine. Atropine more often abolishes the extrasystoles. Dr. Robertson will report the case in full probably after operation. The remaining cases (Nos. 12 to 15), though they are patients with definite cardiac lesions, are included more naturally in Table I, as they were not taken after full digitalis also. They will be referred to later.

Table II.—The capital paper of Silberberg has already been mentioned. He shows the prognostic significance of the atropine reaction in cardiac cases, especially auricular fibrillation, and how it may be used in the study of digitalis action. My cases add nothing new, but are simply fresh illustrations of some of the facts set forth by him. Now let us compare the atropine reaction in some of these fibrillation cases before they are given any digitalis, or any treatment except rest in bed. First of all cases of cardiosclerosis, both those with regular pulse and those developing auricular fibrillation, especially if they have a slow rate, most often give a small reaction to atropine. There are exceptions. Table II, IVa, reaction 18, and Va, reaction 19, are illustrations of the first statement. Table I, No. 12, though his initial pulse was 101, gave a reaction of only 14. Table I, No. 14, whose sclerosis was unmistakable, with no fibrillation, gave the same small reaction, 17 (Fig. 1, *C*). Speaking broadly, it is unusual for cases giving such small reactions to respond well to digitalis, and thus the reaction carries prognostic significance.

On the other hand, patients with rheumatic mitral disease developing auricular fibrillation with rapid pulse rate give large atropine reactions, normal or above (Fig. 1, A). Thus Table II, observations Ia, Ic, IIa, IIIa give reactions of 40, 50, 36, and 28 respectively, that is, practically normal or above. Silberberg had 4 patients who under similar circumstances gave reactions of 44, 53, 60, and 53. Thus we see different cases, or groups of cases, give widely varying reactions under atropine. The pulse rate, the vagal influence being abolished, depends upon the activity of the pace-making part of the heart, that is, it depends upon either stimulus production or conduction. Just which is probably incapable of proof, however, Silberberg's explanation seems more likely. He looks upon the bundle as being highly irritable in these fibrillating hearts with rapid pulse rate, and as carrying an excess of stimuli from the disorderly auricle to the ventricle. Thus a large atropine reaction probably means an irritable bundle, a small reaction some defect in the bundle.

Now let us turn to the role the atropine reaction may play in the study of the action of digitalis on the heart. First of all, let us briefly enumerate a few of the well-recognized facts concerning the therapeutic action of digitalis. Digitalis produces the most decisive slowing of the pulse in those cases of old rheumatic mitral diseases which have developed auricular fibrillation, especially with rapid pulse rate. Digitalis has but little influence on hearts the seat of senile cardiosclerosis, especially with a slow pulse, even though they develop auricular fibrillation.

Patients with a regular rhythm rarely show any such marked response as the first group above. Of course, it is recognized that digitalis may produce a slowing in these cases, but never is the therapeutic effect so brilliant as in the first group. Now the question arises, how does digitalis slow the heart, especially in these cases of auricular fibrillation where the response is so decisive. The possibilities are: Increased vagal inhibition, sinus slowing, depressed conductivity (blocking), and possibly a direct nutritional influence on the whole heart. That is, it must act through the vagus, or on the heart itself, influencing some or all of its functions.

Prof. Cushny suggested that the atropine reaction might be used to eliminate the vagal factor in the digitalis slowing, and thus determine whether it is a large, a constant, or a varying factor. To determine this Silberberg studied 12 cases, mostly auricular fibrillation, in the Mt. Vernon Hospital, and the writer has a few to add.

The patients, having been in the hospital long enough to eliminate the possibility of drug influence from medicines taken before admission, on rest alone, had their atropine reaction taken according to the method described. They were then put on a

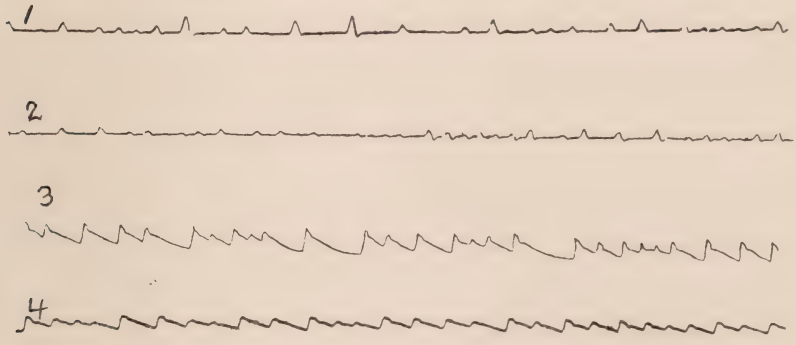

FIG. 2.—Showing character of pulse tracings in auricular fibrillation, Case III, Table II. Unfortunately, unintentionally the tracings of the patients giving larger reactions were destroyed. 1, before any treatment, pulse rate 90; 2, twenty minutes after injection of atropine, grain $\frac{1}{75}$, pulse rate 126; 3, full digitalis (drams 34), pulse rate 55; 4, forty minutes after injection of atropine, grain $\frac{1}{75}$, pulse rate 86; patient under full digitalis.

good preparation of digitalis, usually the tincture, minims 15 to 20, three times a day, and kept on it until they showed their limit of tolerance, anorexia, headache, nausea, and possibly vomiting. Now the reaction was taken again and the results compared.

The group of cases of auricular fibrillation (Table II, Ia, Ic, II, III) show marked slowing under digitalis. A study of the pulse tracings of this group (Fig. 2) shows how the digitalis slowing is produced. Many small beats disappear, the remaining are more even in height, and the diastolic pauses more regular. Turning to the tracings of this same digitalized group when given atropine

we find that many of the small beats reappear at the expense of the diastolic pauses as the pulse accelerates. The type in both instances remains that of auricular fibrillation. The inference is that under digitalis increased vagal influence has prevented many stimuli from getting through. Remove this vagal influence by atropine, and again the fibrillating auricle sends through a rabble of impulses. Again we note in this group under atropine in

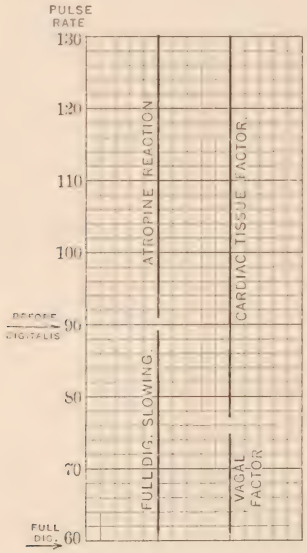

FIG. 3.—Silberberg's diagram adapted to Case I, Table II. The initial pulse before any treatment except rest was 90, and under atropine rose to 130. He was given full digitalis, and his pulse fell to 61. While under full digitalis the reaction was again taken but the pulse rate increased only from 61 to 75, a reaction of 14, which is the vagal factor in the digitalis slowing. Then 130 minus 75 or 55 represents in figures the increment of improvement in the cardiac muscle or the cardiac tissue factor.

digitalis action the pulse rate not only does not reach the height attained under atropine before digitalis was given, but falls short of even the initial rate before any treatment whatsoever. If the vagal factor were the only element in the slowing we should expect to see the atropinized pulse rate reach the same height after digitalis, as it did before. Hence there is evidently a second factor concerned in the slowing, which is probably the

direct result of the digitalis on the heart itself, or the bundle, and is probably nutritional. These factors have been referred to as the vagal factor and the cardiac tissue factor. Their relationship will be most easily understood by referring to Fig. 3. These factors vary in different cases.

The following tabulation will make the matter plainer:

Case.	Ia	Ic	II	III	IV	V	VI
Full vagal effect	40	50	36	28	18	19	30
Full digitalis slowing	29	32	57	36	0	35	0
Vagal factor	14	12	1	29	0	64	40
Cardiac tissue factor	55	70	92	35	18	0	0

In the first 4 cases the tissue factor is by far the greater. This is in accordance with their clinical history, their cardiac tissue was capable of great improvement under digitalis. They are the cases which get well enough to resume at least a part of their ordinary activity. Case II had a bundle most susceptible to digitalis which easily produced blocking. Case IV is typical of the experience with the well developed cardiosclerosis case. They can be promised little improvement under treatment. Case V, a patient in Dr. Sailer's service, showed the average slowing, but it was entirely vagal. He showed no improvement under atropine used therapeutically, nor under digitalis, and succumbed later with dilated heart and pulmonary edema. Case VI was the only post-pneumonic patient available at the time. His sound heart and normal pulse argued that digitalis would have no effect as it proved, but the toxin of pneumonia did not impair his response to atropine as it did in the typhoid cases.

Conclusions. 1. The atropine reaction in the normal heart is probably from 30 to 40.

2. A reaction of 20 or less, in a heart not recently subjected to exhausting disease, points to a degenerative process in the cardiac tissue which makes the outlook for improvement under treatment unpromising.

3. Cases of auricular fibrillation with responses normal or above are promising subjects for treatment.

4. Two atropine reactions in cases of auricular fibrillation, one before and one on full digitalis, enable us to determine whether the vagal or the cardiac tissue factor is the greater. The patients with a large cardiac tissue factor are the ones who usually are sufficiently improved by treatment to return to their occupations.

DISCUSSION

Dr. H. C. Wood, Jr.: A third possible explanation of the slowing of the pulse under digitalis not mentioned by Dr. Talley is the fact that digitalis acts directly upon the ventricular muscle. It is not impossible that the slowing of the pulse is due to a prolongation of the systole rather than to an influence upon the conductivity.

Dr. H. A. Hare: I believe the subject is an exceedingly important one. There is no doubt that if atropine is used in proper cases and in suitable dosage, in many instances of cardiac disease due to organic change, it will prove as valuable as digitalis. Another point that I should like to speak of is that most of us are a little afraid of atropine. I have been using a twenty-fifth of atropine with gratifying effects. I believe that in many cases of great cardiac irregularity where we are prone to think that perhaps the lesion lies in the heart muscle we will find some disturbance of the vagus. Atropine will often clear up this condition.

Dr. Talley closes: When the use of one-fiftieth to one twenty-fifth of a grain of atropine hypodermically was first recommended in hemoptysis I felt a little afraid of the dosage. However, the results appeared good in large and persistent attacks of hemoptysis. During the last year I have made literally dozens of such injections of atropine in taking this reaction and have had no marked complaints. Of course, there is usually dryness of the throat and some dilatation of the pupils, but the patient is ready for the next meal, sleeps well, and reads the paper as usual in a few hours.

THE CEREBRAL MECHANISM OF EMOTIONAL EXPRESSION[1]

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LITERATURE

Emotion and emotional expression can be approached from several standpoints, as from biology, anatomy, physiology, psychology, pathology, and physiognomy. The organic basis of emotional expression in man, which will be in considerable part the subject of the present contribution, includes the neural and muscular periphery, the structural correlatives, spinal and bulbar, of this periphery, and the controlling and regulating cerebral and cerebellar, cortical and subcortical centres and tracts concerned. While central mechanisms will in the main be discussed attention will be directed to other elementary features of the subject in order to take a comprehensive glance at the whole.

No attempt will be made to present a complete study of the literature of emotion and emotional expression, for which the reader is referred for enlightenment on different phases of the subject to such well-known authors as Sir Charles Bell, Charles Darwin, Hughlings Jackson,[2] and the school of localizationists,

[1] Read, March 6, 1912. This paper as presented at the College of Physicians was illustrated by numerous lantern slides and moving pictures, a list of which is appended to the article. The moving pictures which were exhibited under the direction of Dr. T. H. Weisenburg include cases considered in the paper and others chosen for the purpose of general demonstration.

[2] Besides Hughlings Jackson, the school of localizationists embraces many names like those of Hitzig, Ferrier, Charcot, Horsley, Dana, and so many others whose work is well known that no specific reference need be made to any. In addition to the literature referred to in the article a partial bibliography is appended.

James, Irons, Wundt, Lavater, Mantegazza, von Bechterew, Nothnagel, von Monakow, Brissaud, Lewandowsky, Obersteiner, Mingazzini, and Bianchi. In the elucidation of the phenomena of emotional expression the original work in artistic anatomy of Dr. George McClellan, of Philadelphia, holds a place of first importance.

Among my immediate colleagues excellent work has been done by Spiller, Lloyd, and Weisenburg, on the subject of the central disorders of emotional expression, especially in articles on pseudo-bulbar paralysis and diseases of the great basal ganglia.

In 1907 Dr. Spiller collaborated with me in the production of an article on the symptomatology of lesions of the lenticular zone, in which the pathology of involuntary laughing and weeping was incidentally considered.

PARALYSIS OF EMOTIONAL EXPRESSION IN MAN

The clinical history of a case in which emotional paralysis of one side of the face was the striking feature was recorded by me in 1908. This patient died in October, 1911, and in February of the present year I published a preliminary note on a new symptom-complex which included emotional paralysis of one side of the face, this contribution containing an abstract of the microscopic findings and a *résumé* of the clinical history of this case. It is only necessary to repeat here that the features of the syndrome were marked ataxia of the upper and lower extremities on the left side, with paralysis of emotional expression in the face, nerve deafness, and loss of the sense of pain, heat, and cold on the right. The pathological examination showed destruction of the ventral portion of the dentate nucleus and adjacent superior cerebellar peduncle. The upper two-thirds of this peduncle were degenerated, and as a consequence the right nucleus ruber was at least one-third smaller than the left. The left mesencephalic root of the fifth nerve and left lateral lemniscus were also much degenerated.

The cause of the loss of emotional expression in the face, while

as yet not thoroughly determined, would appear to be related to the destruction of afferent tracts in the lemniscus, or of this and some part of an important cerebellar arc.

Recorded examples of emotional paralysis like the above are rare, the reason for this being twofold. In the first place, such loss of expression either in the face or limbs or in both is in fact infrequent as the result of disease, and especially disease of the central nervous system; and secondly, this form of impairment or loss of control is doubtless at times overlooked, the investigator's attention being usually confined to a study of voluntary movement.

Spiller has published with photographs a case in which partial emotional paralysis on the same side was present, the case being one of tumor of the cerebellopontile angle. He inclines to the view that the partial emotional palsy was dependent upon interference with the cerebellar arc by way of the posterior cerebellar peduncle and dentate nucleus. Two cases reported by Nothnagel are well known, although owing to the size of the lesions which involved various parts besides the thalamus and adjoining regions, the inference as to a thalamic centre of mimetic expression, according to the views of von Bechterew is not as sure as Nothnagel and others who have made use of these cases believe. While the thalamus doubtless plays a part on the afferent side of the bulbocerebral mechanism of expression we are not yet in position to decide as to the exact expressional role of this ganglion, but this subject will be considered somewhat more fully later in the paper.

EMOTIONAL EXPRESSION IN LOWER ANIMALS AND IN MAN

The muscles of expression in the face of the lower animals differ considerably from those of man, both in accordance with the general form of the animal's head and face and with its power of emotional expression. Carnivorous and graminivorous animals, as Bell has shown, differ considerably in their facial musculature. In the former these differences are such as to

make special variations in expression, particularly as indicated in showing the teeth and in the use of the eyes.

New muscles in the face are added as animals rise higher and higher in the scale. This is illustrated by a comparison of the anthropoids and man with the commoner carnivorous and graminivorous animals. Among the added muscles are those particularly concerned in expression, as the corrugator supercilii and the triangularis oris. In the anthropoids these muscles while present are not as finely developed for the expression of emotion as in man.

For the purpose of the present discussion it must be constantly borne in mind that the nervous mechanism of emotional expression in the lower animals is less independent of the will and shows marked points of difference from the nervous mechanism of emotion in the human being. Emotion as apparently expressed by these animals is usually more immediately related with what they are doing or are about to do. While it may be instinctive or reflex it is not conditioned as much as in man by the presence of an independent emotional system.

EMOTIVE CORTICAL ZONE (MIDFRONTAL OR PREFRONTAL)

In the early years of the modern era of cerebral localization it was held that the motor zone of the cortex extended for an equal or almost equal distance, both cephalad and caudad of the central fissure. As time elapsed, however, experimentation upon the lower animals, more especially upon the anthropoids, and the more careful observation and interpretation of clinico-pathological phenomena, demonstrated that this area was largely, if not altogether, in front of the Rolandic fissure. As shown in the scheme by the writer, first published in 1905, in a paper written in conjunction with Dr. Frazier, it will be seen that the motor zone thus represented is largely confined to the precentral convolution, having, however, a projection or extension forward into the second, and superior portion of the third frontal convolution. In the anthropoids it is even more confined to the precentral convolution than in man.

As animals have ascended in the scale, new centres of representation have developed as evolutional outgrowths of the original motor zone. In the case of the human being such development has always taken place as man has added to the movements necessary for existence, those especially related to acts of skill and emotional expression. Such development has been toward the anterior pole of the brain. Centres for writing and for speech are in part the result of this forward expansion, but with these I am not particularly concerned in the present communication.

In lower animals, as has been so beautifully demonstrated by Darwin, emotion is expressed by movements of various parts of the body, although the face is not entirely excluded from such manifestations. In man facial movements are preëminently the means of expression, and cortical centres concerned with the representation of movements of expression must exist, as such expression is not always involuntary, and even when known to be non-volitional, has had recently or remotely a voluntary origin.

Experimental faradization of the human midfrontal cortex during the performance of craniectomies of considerable size have shown that the movements evoked are such as are commonly used in facial emotional expression, although except in a few instances not confined to the purposes of expression.

Some of the movements obtained by such electrical excitation were those of turning the head to one side, of forward bending of the head, of closure of both eyes (other observers having noted opening of both eyes), and of opening and shutting movements of the jaws and mouth. The results thus obtained were from the faradization of cortical regions in advance, and some of them much in advance, of the precentral convolution, in which most of the unilaterally differentiated movements are represented as has been indicated by various methods of investigation. Movements of the jaw and of opening the mouth seem to have a large area of representation well forward in the motor region.

Recently, during the search for a tumor, the frontal lobe from the central fissure to within about one to two inches of the anterior pole of the brain was exposed by Dr. Frazier. Unipolar faradiza-

tion at a point in the second frontal convolution, near the junction of its middle with its anterior third, caused elevation slightly toward the median line of the inner angle of the eyebrow on the side of stimulation and elevation of the eyelid. During the same operation stimulation farther back caused depression of the angle of the mouth and twitching of the platysma on the opposite side. Movements of the eyebrows and eyelids are a part of those concerned with the expression of grief.

It may be interesting to observe here in connection with the effect obtained of elevating one eyebrow by stimulating the prefrontal cortex of the same side that an individual occasionally has an unusual facility in the volitional performance of this movement. A well-known Russian actress sometimes makes use of this movement in changing her facial expression. The movement in the human being seems to be epsilateral as demonstrated by our experiment, but it may also be a contralateral movement or a part of one that is bilateral. Many of the movements of expression are of course as is well known bilateral, the centre of one side governing the movements of both.

The centre for the corrugator supercilii is doubtless situated in the immediate vicinity of the point, faradization of which resulted in elevation of the inner angle of the eyebrow. The movements of the face which express distress, pain, or anguish are of late development in the process of evolution and probably have their centres of representation situated in advance of all other motor centres.

In these experiments I was searching for the centres for the movements of what Darwin has aptly called the grief muscle, believing these on theoretical grounds to be in the most anterior portion of the motor zone, or more probably cephalad of this zone as usually described. Darwin describes the muscles which give the peculiar appearance of the central portion of the face between and above the eyes as due to the contraction of the orbiculars of the eyelids, the pyramidals of the nose and the corrugator. Darwin holds that the movements of these muscles are apparently checked by the more powerful action of the central

fasciæ of the frontal muscle. The movement due to this fascia on the side of application was the one obtained in our experiment.

Owing to the exigencies of the operation it was deemed best not to increase or continue the stimulation, but I have no doubt if this had been done in the same vicinity the movements of the corrugator supercilii, orbicularis palpebrarum, pyramidalis nasi, and the frontalis would have been obtained on both sides. In other words, in the region where fragments of this general movement were determined the whole could have been obtained by sufficient stimulation.

Duchenne, who is cited by Darwin, differs somewhat from the latter, believing that it is the corrugator which raises the inner corners of the eyebrows and that this is antagonistic to the upper and inner part of the orbicular muscle as well as to the pyramidalis nasi. It is likely that it may eventually be found that centres for all the central and bilateral movements of the face concerned with expression are located in what we have chosen to term the emotive zone or area.

The part played by the posterior extremity of the third frontal convolution in speech has interest in this connection, as the first attempts at speech by primitive man were probably in response to emotions called out by pain, hunger, fear, or other causes.

HISTRIONIC AND SPONTANEOUS LAUGHING AND WEEPING AND OTHER ALLIED FORMS OF EMOTIONAL EXPRESSION

The spontaneous movements of laughing or weeping are regarded as among the most automatic, instinctive, or reflex of the movements of expression. The mechanism for voluntary movements seems to have but little share in the phenomena. The ability voluntarily to imitate laughing, weeping, frowning, and other more or less marked expressions of emotion, without or with the presence of such emotion, must be brought about through some association between the voluntary and involuntary mechanisms. Borée, an Alsatian actor, was photographed in many poses intended to indicate the different emotions. These were collected

in a volume of 119 autotypes, representing crying, care or anxiety, and laughing, etc. One thing which seems evident in making a comparison of histrionic and spontaneous emotional expression is that the former has a forced element not present in the latter, in which an appearance of relaxation predominates.

With Dr. Weisenburg, with the aid of moving pictures, I studied the simulation of various emotions by well-trained actors, comparing the expressions produced with those exhibited in spontaneous laughing and weeping as illustrated by actual cases and by photographic reproductions of Darwin and others. However, skilful the reproduction a difference between the two manifestations is more or less apparent, a difference which is probably determined in large degree by the manner in which the voluntary motor mechanism is associated or dissociated from that for so-called involuntary movements. Whether emotional expression is ever absolutely divorced from voluntary movement may be doubted, its discussion involving perhaps some consideration of the theories which hold that movement precedes emotion, or the reverse.

EFFECTS OF MUTILATION OR DISEASE ON THE MUSCLES OF EXPRESSION

The brutal but skilful barber surgeon of the time of James II cut the muscles of the face in young children in such a way that while the entire musculature might be at rest momentarily, or even for a longer time under the strong influence of the will, the victim of this procedure was always in the imminence of grinning or wild laughter. This mutilation was in the interest of the cruel trade of supplying involuntary laughers for mountebank performances. In a case of this sort the factitious peripheral state is one in which involuntary expression could not be sufficiently controlled by the cerebral inhibitory mechanism, hence the explosions of facial movement. That the muscles under the conditions present be controlled at all, proves the existence of an ordinarily strong and efficient inhibitory nervous apparatus.

It is an interesting point in connection with cases of irrepressible laughing and weeping of different sort that the facial innervation, and probably the innervation of other parts of the body concerned with the expression of emotion, is often weakened in the intervals between the involuntary outbreaks. I have again and again noticed this, although it is not manifest in all cases. The masked appearance of the countenance spoken of by von Monakow is sometimes in evidence as in one of the most striking instances which I have ever seen of irrepressible weeping associated with howling. This man's face when at rest reminds one of a countenance void of emotion, a dull or blank expression characterizing it. The movements in these cases are bilateral as is also the masking of the features between the outbreaks. As von Monakow well puts it the irrepressible agitation gives the impression of a ready mechanism which in normals is regulated by inhibition, finely controlled by the will, which is suddenly freed from all fetters and breaks roughly into prominence. When the musculature was ingeniously cut, in Hugo's *Man Who Laughs*, so as to put it, so to speak, as if on a knife-edge balance, the powerful internal inhibitory mechanism could not be continuously effective.

SUMMARY OF THE SITES OF ORGANIC LESIONS CAUSING DISORDERS OF EMOTIONAL EXPRESSION

Lesions very diversely situated may produce loss, exaltation, or disorder of emotional expression. Pathological evidence shows that such lesions are usually, but not always, bilateral. When unilateral, they are most frequently right-sided. These lesions may be situated (1) in the emotive cerebral cortex, or jointly in this and the motor zone as usually recognized, in which case they are bilateral or one-sided, and when unilateral, large and either very destructive or very irritative. (2) Bilateral lesions of the frontothalamic tracts (?) (3) Bilateral lesions of the frontopontile tract. (4) Bilateral lesions of the lenticula. (5) Bilateral lesions of the geniculate or corticobulbar tract of the internal capsule. Sometimes lenticular and capsular lesions are present

in the same case. (6) Bilateral lesions of the thalamus (von Monakow and Nothnagel) (?) (7) Bilateral or right-sided unilateral lesions of the red nucleus and cerebral peduncle. (8) Bilateral lesions or central or unilateral lesions of the pons. (9) Unilateral or bilateral lesions of the dentate nucleus and superior cerebellar peduncle and lateral lemniscus.

EMOTION AND EMOTIONAL EXPRESSION AND THEIR CORTICAL REPRESENTATION

A word or two might first be said here with regard to the mental process called emotion and some of the theories which have been advanced regarding its cerebral structural basis.

No sharp distinction can be made as some would have us believe between feeling and emotion. They are to our way of thinking but degrees of the same general process. According to the James-Lange theory of emotion the mental process so denominated succeeds rather than antedates the movements which usually express emotion, the sequence being perception or sensation, movement and emotion. If this is the case, the question of emotional expression becomes more important in the consideration of the nature and probable seat of emotion.

This question of the probable seat of the representation of emotion (and here I am not speaking of emotional expression) has not escaped the interest and attention of some neuropsychologists. According to Flechsig, for instance, the general sensorimotor cortex is the central focus or seat of the emotions and passions. He accepts von Bechterew's view of a thalamic centre for emotional expression, and believes that impulses from the sensorimotor cortex, as understood by him, are conveyed to this thalamic centre and thence to the periphery. Some have held that in the gyrus fornicatus, others that in the post-central convolution, and still others that in the thalamus itself painful sensations are represented.

An American psychologist, Mary Whiton Calkins, has attempted to formulate a theory of the localization of pleasure

and pain. This will be best understood by giving it in the language of the writer in the citations which follow. The views which are apparently accepted by Calkins with regard to sensorimotor localization are not those held by me, as I hold to the separate representation of sensation and of movement in the cortex, and also that the motor area is entirely or almost entirely cephalad of the central fissure. This difference of opinion, however, does not prevent a comprehension of the hypothesis advanced as to the brain structures concerned in emotion and in its expression.

"Pleasantness and unpleasantness," says Calkins, are occasioned by the excitation of fresh or of fatigued cells in the frontal lobes of the brain, and the frontal lobe is excited by way of neurones from the Rolandic area of the brain. When the neurones (or cells) of the frontal lobes, because of their well-nourished and unfatigued condition, react more adequately to the excitation which is conveyed to them from the Rolandic area, an experience of pleasantness occurs; when, on the other hand, the cells of the frontal lobe, because they are ill nourished and exhausted, react inadequately to the excitation from the Rolandic area, then the affection is of unpleasantness; when, finally, the activity of frontal lobe cells corresponds exactly to that of the excitation, the given experience is neither pleasant nor unpleasant, but indifferent."

"But we have not completed our study of the bodily conditions of emotion," says this writer again, "until we try to discover the brain or nerve changes which condition these changes in pulse, respiration, and the rest. A probable account of these brain changes is the following. First (*a*) sensory brain centres are excited through perception or imagination of a given object; next (*b*) the excitation of these sensory neurones spreads to the brain centres of bodily sensations and movements, that is, to the region forward and back of the fissure of Rolando, and there excites motor cells. This excitation of the motor neurones of the Rolandic region is then carried (1) downward to lower brain centres in the *medulla oblongata*, which control the unstriped muscular coatings of the inner organs of the body, such as bloodvessels, heart, and intestines. In this way

the internal circulatory changes are brought about: the heart-beat and pulses are checked or increased, and the arteries (not the big ones near the heart, but the smaller, thin-walled vessels in outlying parts of the body) are dilated or constricted, thus occasioning either a flush and rising temperature or pallor and chilliness. The downward excitation is carried (2) to the striped or skeletal muscles attached to the bones of the body, and thus the 'external' changes in breathing and muscular contraction are occasioned. Both sorts of bodily change, the 'internal' and the 'external,' excite end-organs of pressure, and the internal changes excite also end-organs of warmth and cold; and these excitations of the end-organs of pressure and of warmth or cold are carried upward by ingoing nerves to the sensory cells of the bodily-sensation-and-movement-centre (the Rolandic area). The excitation of these sensory cells is the immediate condition of all the organic sensations (whether due to internal changes or to external movements) which are present in emotional experience. And from the Rolandic area, excitations carried to the frontal lobe bring about that adequate (or inadequate) excitation of neurones which conditions the pleasantness (or unpleasantness) of emotions."

If either of these theories, and especially that of Calkins, be tentatively accepted, and if in addition we adopt the James-Lange theory of emotion following rather than preceding the muscular movements, in which are to be included not only the movements of skeletal muscles but also the movements of circulation, respiration, secretion, excretion, etc.—by which emotion is expressed, it would seem logical to believe that the cortical representation of emotional expression is closely related in the cerebral mantle to the cellular region of the brain in which the activities which more particularly cause emotion are themselves represented. If instead of the James-Lange theory we adopt that of Irons and many others who believe that movements follow or are closely concomitant with emotion, the same would probably be true with regard to the cortical representation of emotional expression.

I am not one of those who believe that the problem of emotion or of any other great mental process is to be explained by regarding it in some vague way as a complex expression of the action of the cerebral cortex as a whole. It may be true that the multitudinous centres, receptive, associative, ideational and emissive, take part in a general sense in all those processes which we collectively regard as mental or mind products. Nevertheless, the trend of all modern investigation as to cerebral function is in the direction of developing a belief in the centralizing or polarizing in certain more or less limited areas of the cortex the particular activities which are concerned with lower and higher mental processes—with concrete and abstract thinking.

With Bianchi and others I believe that in the frontal lobes, especially in the prefrontal region, occurs the final synthesis of those cerebral activities which result in the forms of conscious action or recognition which are included under such terms as attention, judgment, comparison, fancy, and emotion. It may be that some of these processes, as attention for instance, have also lower or special representation in centres and associative tracts more directly connected with the periphery, but a part, and a higher part, of such processes has its synthesis in this frontal or prefrontal region.

Where then is the site of the expression of emotion? A full consideration of all the phenomena associated with emotion—those of so-called spontaneous laughing and weeping in children or adults, of consciously forced emotion or emotional expression, as in acting, of such expression as the result of ideation in itself dependent upon memorial residua—cannot but lead us with our knowledge of function to the opinion that the cortex or cerebral mantle has always a place in the explanation of the phenomena. It may be urged that involuntary or spasmodic laughter or weeping such as has been so well observed and studied by numerous neurological investigators, occurring as it most frequently does because of lesions in subcortical centres and tracts, such as has been previously summarized, indicates that the representation of emotional expression is in these parts. But in reply to this it may be said that we have here to consider not true emotion or its repre-

sentation, but the action of parts concerned subcortically, that is, unconsciously, with the expression of what appears to be emotion. It is a frequent observation by those who have studied cases of these forms of disease that the patients apparently exhibit these phenomena of involuntary laughing and weeping without any concomitant emotion, or if emotion be present it is a sequence rather than a concomitant or precedent of the involuntary emotional expression. These submantellar areas and tracts, it can also be demonstrated, have structural connections with definite portions of the cortex and especially with frontal areas in which it is universally believed are inhibitory centres or mechanisms, and also, it is believed by some, psycho-excitory centres or mechanisms.

Briefly stated, my views with regard to the cortical representation of emotional expression are that in the midfrontal region of the cerebral hemispheres, and especially on the right side, is an area which may be regarded either as an independent zone or an anterior subdivision of the general motor zone. Here are cortically represented those movements which are especially concerned with the expression of emotion. The term *movement* in this connection is given a broad significance as applying not only to the movements performed by skeletal muscles, as the movements of the face and limbs, but to movements visceral, vascular, secretory, in fact all movements which result in any way in adding to the sum total of emotional expression. Let me say here again that I am not now speaking of areas and centres of emotion, but of emotional expression. That part of the frontal lobe which takes a chief part in the special synthesis which express themselves as the mental process called emotion, is in all probability closely contiguous to this zone of emotional expression. The cortical area of emotional expression, in other words, is intercalated between the motor zone as usually described and the great prefrontal psychic sphere. Perhaps the last interlaces in its structural arrangement with this zone of expressional representation.

This view is not as extreme or as improbable as might to some at first sight appear. The more primitive zone of motor repre-

sentation in the left hemicerebrum has anterior to it an evolutional outgrowth which corresponds to the cortical motor centre for speech and cortical motor centre for writing. As evolution advances cerebral development takes place in a caudocephalic direction. As the skilled movements of speech and those necessary for writing and drawing have been added to the human equipment, this midfrontal region has more and more attained importance.

Similarly in the cerebral hemispheres, and especially in the right cerebral hemisphere, anterior to the more primitive motor zone has been developed a zone for the representation of emotional expression. Perhaps it may not be spoken of as a region for the representation of skilled movements, or at least this idea is not so apparent as in the case of speech and writing; nevertheless emotional expression correctly regarded holds a higher place than the representation of voluntary movement.

Earlier in this paper I have presented some of my reasons, drawn from faradization of midfrontal and prefrontal cortical areas for regarding this zone of emotional expression as located in front of the motor zone as ordinarily regarded. Some clinicopathological evidence in favor of this hypothesis has also been collected by me. The case, with the smiling or laughing aura, a history of which is appended, being in this category.

In this connection I would call attention to the fact that Nothnagel in discussing his often cited cases of mimetic facial paralysis, so commonly used to uphold the hypothesis of von Bechterew of the thalamic centre for mimetic expression, remarks that further experience may lead to the conclusion that somewhere in the cerebral cortex this emotional innervation may have a definite place, a zone or area which is the rallying point for the psychic reflexes. Impulses arriving by the various tracts of the cortical association systems being gathered here before passing into the tracts through which impulses concerned in emotional expression are discharged. This rendezvous, if it exists, would be the cortical emotive zone suggested by me as located in the midfrontal, or midfrontal and prefrontal cortex.

THE PART PLAYED BY THE THALAMUS AND OTHER BASAL GANGLIA IN THE MECHANISM OF EMOTIONAL EXPRESSION

The thalamus, as has already been indicated more than once in this presentation, has long been supposed to play an important if not a dominant role in emotional expression. Many of the earlier neurophysiologists regarded the thalamus as a motor organ, but even by some of those who had largely disregarded this view of its functions as a whole, some portion of it continued to be looked upon as motor or efferent. Von Bechterew in particular taught that in the thalamus, especially in its medial portion, is a centre for mimetic expression, irritation or destruction of which on one or both sides would cause on the one hand increase in emotional movements or on the other hand paralysis of such movements. This hypothesis of von Bechterew has been adopted by many, and it has seemed to me, as to some others, without adequate experimental or clinicopathological evidence. The main data, physiological and clinicopathological, as given by von Bechterew on which his view is founded, can be summarized about as follows:

(1) If all parts of the brain down to the thalami are removed, the animal loses, along with the psychic functions, its power of voluntary motion; but it retains, along with the simple reflexes, all the movements of expression; among other phenomena many kinds of vocal utterances occur reflexively such as can be evoked in normal animals also. (2) If the cerebral hemispheres together with the thalami are removed the animal loses, along with its psychic functions, the ability to perform all the complex movements of expression; there remain only the simple motor phenomena of a general or local character, like restlessness in pain, as well as reflexive vocal utterances, which in this case are evoked only by the painful irritation, and tend to become more or less monotonous. (3) If one irritate the thalami by penetrating their mass with an electric needle, particularly in their distal part, certain movements of expression are produced, as well as

many kinds of vocal utterances (such as growling, baying, or howling in dogs), which appear the more readily upon bilateral irritation. (4) If one destroys or injures in animals the thalami in their posterior parts, leaving the cerebral hemispheres intact, the power for movements of expression will be more or less modified, along with the remaining motility; indeed, in individual cases it amounts to a mimetic paralysis of the facial muscles while the power of voluntary motion is retained. In the case of a one-sided lesion in the interior of the posterior region of the thalamus, this mimetic facial paralysis with preservation of voluntary movements occurs on the opposite side.

Von Bechterew believes that he has confirmed his experimental investigations and the views based upon them, by personal clinical observations and also by the collection of clinical and clinicopathological observations of others.

On a careful consideration of this exposition of the views of von Bechterew one can readily accept all the facts as given without necessarily concluding that the thalamus is to be regarded as in any of its parts a motor centre. Interference with its functions as a great station on the afferent side of the nervous system might cause just such phenomena as are described by von Bechterew and used by him in upholding his theory of a special motor (mimetic) thalamic centre. Before, however, considering this question from the standpoint of the recent observations and theories regarding the functions of the thalamus, and especially those of Dejerine and Roussy and of Head and Holmes, brief reference might be made to a few of the many neurologists and physiologists of high authority who have supported the stand taken by von Bechterew and also to the views of those who have held that the mimetic centre was placed elsewhere, as in the lenticula.

Nothnagel in discussing his cases of one-sided mimetic facial paralysis adopts the view of von Bechterew with regard to the part played by the thalamus as a psycho reflex centre for emotional expression. One of his cases, however, because of the size of the lesion which involved much more than the thalamus, does

not appear to be conclusive in support of the theory. He clearly shows, however, as others have shown, that in order to explain the phenomena both of emotional paralysis and emotional over-action it is necessary, as regards centres, tracts, and associations, that the mechanism of voluntary movement and that of emotional expression should be largely independent of each other.

Brissaud in one of his instructive and entertaining lectures, in which he discusses involuntary or spasmodic laughing and weeping, seems to adopt the hypothesis of von Bechterew as regards a thalamic mimetic centre, with some special views of his own as to its relations to the cortex and other parts. He shows the evident part played by the bulbar nuclei in emotional expression.

He also shows what is well known, that lesions variously situated as in the bulb, in the posterior or anterior limb of the internal capsule, or in the thalamus may be the pathological cause of the involuntary emotional outbursts. He especially emphasizes the fact that bilateral paralyzing lesions of the voluntary motor bundle in the internal capsule may cause spasmodic laughter or weeping, giving one of the pseudobulbar forms of this affection; and also, he holds that bilateral or perhaps unilateral irritative lesions of the corticothalamic bundles may give the same if the excitation is sufficient.

Brissaud while apparently adopting the views of von Bechterew at the same time criticises them. He cannot see, as others have not been able to see, how destructive lesions of the thalamus would cause the effect attributed to them by von Bechterew unless the lesions were small and partly of an irritating character.

As indicating the widely separated lesions which may cause involuntary laughing and weeping, and therefore the elaborate extent and distribution of the structural basis of emotional expression, I might refer to the fact that Brissaud cites cases of hemiplegia or hemiparesis, of disseminated sclerosis, of amyotrophic lateral sclerosis, of bilateral primary lateral sclerosis, of apoplectic injury causing paralysis of all four extremities, of diseases due to encephalitis, of simple atrophy of the brain, and of porencephaly.

Brissaud speaks of the important part played by what it seems he first designated, when studying under Charcot, the "psychic bundle," by which is to be understood the fasciculus which passes by way of the anterior limb of the internal capsule from the frontal lobe to the thalamus.

Instead of the thalamus the lenticula has been supposed by some to contain centres for mimetic expression on the efferent side of the nervous mechanism, a view which while not proved has as much in its support as the von Bechterew-Nothnagel thalamic hypothesis. Mingazzini holds that the thalamic fibers probably pass by way of the putamen of the lenticula, believing also that the voluntary geniculate bundle takes part in inhibition. Goldstein appears to maintain a view similar to that of Mingazzini as he refers spasmodic laughing and weeping in some cases at least to lesions of the lenticula.

The weight of recent evidence as well as much that has been learned in the recent or more or less remote past, strengthens the view that the thalamus is an organ entirely on the afferent side of the nervous system. Dejerine and Roussy and Head and Holmes have contributed much to emphasize this position and also to elucidate the functions of the thalamus and the clinical phenomena produced by its disease. All afferent fibers passing upward from the midbrain appear to end in this ganglion, and here, using the language of Head and Holmes, "Lie the synaptic junctions of those paths by which impulses are carried onward to the cortex." I shall not here discuss the thalamic syndrome of the authors just cited, but merely call attention to a few points which seem to bear upon the discussion of the question of the mechanism of emotional expression.

In passing, I might refer to the statement of Malone and Jacobsohn, cited by Lewandowsky, that there is in the thalamus no single cell of motor structure. Lewandowsky's views, as indicated in his discussion of the centripetal, centrifugal, and associating cerebral tracts, are opposed to that of von Bechterew and his followers.

Lewandowsky describes the many connections of the cerebrum with the pontile nuclei, which connections may prove significant

in the final elucidation of the question of the efferent side of the emotional motor mechanism.

In a paper like the present, not so much intended for neurologists and neuropathologists as for those generally interested in the question of emotion and emotional expression, it will not be necessary for me to describe in detail the many connections, centripetal and centrifugal, cordicofugal and cordicopetal, of the thalamus. The work of many neuropathologists, like von Monakow, Obersteiner, Lewandowsky, Holmes, and Spiller, has contributed to our exact knowledge of the elaborate structural relations of the thalamus with other parts — with afferent tegmental tracts, with paths from the cerebellum by way of the nucleus ruber, with to-and-fro connections to all parts of the sensory cerebral cortex, with the frontal region, with the caudatum and with the putamen of the lenticula. What is necessary to be understood is that the thalamus, a great station on the afferent pathway, is so situated as to play a distinctive role in emotion and emotional expression. Painful and pleasureable stimuli are here received, interaction between the sensory cortex and the thalamus taking place in such a way as to bring about a clearer recognition and discrimination of the sensations received, while the frontal cortex acts in an inhibitory or regulating manner on the thalamic centre for sensation, and the cerebellum gives the necessary tone and coördination.

Let me cite here from Head and Holmes an instructive paragraph.

"The anatomical structure known as the optic thalamus is an extremely complex portion of the brain, and contains not only the terminal centre for certain aspects of sensation, but plays a three-fold part in the fate of sensory impulses:

"1. It contains the termination of all secondary paths; here sensory impulses are grouped afresh and redistributed in two directions, on the one hand to the cortex cerebri, and on the other to the gray matter of the optic thalamus itself.

"2. It contains a mass of gray matter, the essential organ of the thalamus, which forms the centre for certain fundamental

elements of sensation. It is complementary to the sensory cortex and exercises different functions in the production of sensation.

"3. The lateral part of the optic thalamus is the organ through which the cortex can influence the essential thalamic centre, controlling and checking its activity. The excessive response to affective stimuli, so prominent a feature of lesions in this situation, is not due to irritation but to removal of cortical control."

Excessive response to affective stimuli, painful and pleasurable, is a notable feature in the thalamic syndrome of Roussy as described by him and by Head and Holmes. Some of the cases recorded by these authors have much interest in connection with this subject of emotion and emotional expression and their organic basis. Head and Holmes, for example, speak of two cases in which music produced not only disesthesia or distressing sensations on the side rendered anesthetic by the thalamic lesion, but even choreoid and athetoid movements of the limbs of this side.

EMOTION AND THE RIGHT CEREBRAL HEMISPHERE

I have spoken of the zone of emotion and emotional expression as especially developed in the right cerebral hemisphere. It is not a new idea that the right hemicerebrum plays a larger part in the realization, control, and excitation of emotions than the left. Among those who early discussed this subject was E. C. Seguin, of New York, who believed from his personal observations that emotional expression was most disturbed in cases of right-sided cerebral disease, and, therefore, of course of left-sided paralysis.

Others have contributed now and then to the exposition of this same idea. I have had the matter under observation for many years. Interesting cases of explosive laughter associated with left hemiplegia have fallen under my observation. Usually when involuntary and spasmodic laughter and weeping are the result of a unilateral lesion, that lesion is somewhere in the right half of the brain. It may be a large cortical or subcortical destructive lesion, or it may be, as in one case which I have the opportunity of pre-

senting in this paper through the courtesy of Dr. Weisenburg, a case of lesion of the right ruber and adjoining cerebral peduncle. It may in fact be a lesion anywhere situated in or below the cortex before decussation of the motor pathways.

Emotional representation would seem in some respects to take the place in the right hemisphere which is held by language representation in the left. In the cortex of the right hemisphere are probably more highly developed special centres for the control of the bilateral or median movements concerned chiefly with the expressions of emotion.

Both eyebrows, both eyelids, both eyes, both sides of the face, the limbs of both sides take part in emotional expression, and the representation of their movements must, of course, be in correspondence with this fact. Emotion as evidenced by blushing or pallor spreads itself over the entire face or it may be over the entire trunk and limbs. What might be classed as the median or centrally placed movements of expression are really bilateral and represented in each hemisphere, but especially in the right. Such movements are, for example, as has already been discussed, those of the corrugator supercilii and the muscles which act with it in the expression of grief. The movements of opening the mouth, might be placed in the same category, while depression of the corners of the mouth may be a unilateral but is most frequently a bilateral movement. Pupillary movements also are of this kind when expressive of emotion.

SOME CASES ILLUSTRATIVE OF DISORDERS OF EMOTIONAL EXPRESSION FROM CEREBRAL DISEASE

My purpose in this contribution has been to present the subject of the mechanism of emotional expression in a general way with lantern slide illustrations to emphasize and add interest. From a large experience I could of course bring many cases of organic nervous disease illustrating special forms and phases of emotional disorder, but this would extend the paper too much. My personal experience includes cases of emotional disorder from pseudobulbar paralysis, from disseminated and other forms

of sclerosis, from deep-seated lesions of the pons oblongata, from lesions of the lenticula and of the peduncles, cerebellar and cerebral, and from large or disseminated lesions of the cerebrum itself. These experiences simply confirm the observations of others as summarized when speaking of the theories of the encephalic mechanism of emotion and emotional expression, observations which show how widely separated are the lesions which may cause disruption of this mechanism, and, therefore, how elaborate the structural bases of this mechanism must be. In concluding the paper I shall simply present the details of several hitherto unpublished cases illustrating a few features of the subjects discussed.

Case I.—*Apoplectic stroke causing left hemiplegia with right third nerve involvement; slight impairment of sensation and decided ataxic tremor on the left; spells of uncontrollable explosive laughter.*

M. M., white, married, a machinist, aged forty-seven years, was admitted to the Philadelphia General Hospital on July 10, 1906, this being his second admission, the first having been in February, 1906.

The following notes were taken just after his admission. He had used alcohol freely. He had a history of chancre twenty years before coming under observation; otherwise he was well until in 1889 he had an apoplectic stroke which paralyzed him in the left arm, leg, and face. The patient said that on the night of the stroke he had a sensation of pins and needles all over the body, and imagined that somebody was playing a practical joke on him with electricity. He arose and looked under the bed for the battery. From this time on he gradually became unconscious and remained so for two days. The stroke confined him to bed for about a year. He had diplopia and right-sided ptosis, which also lasted about one year. The paralysis, both of the left side and of the right third nerve, gradually improved.

The pupils were equal and reacted to light and accommodation. Associated movements were preserved. The nasolabial fold on each side was normal. He had equal control of the muscles on each side of the face. The power of the muscles of mastication was the same on each side. Cutaneous sensation was a little diminished on the left. The grip was not quite so strong on the left as on the right side. No atrophy of the muscles of either arm was present. The triceps and biceps tendon reflexes were more pronounced on the left side than on the right, though they were not much exaggerated.

On the left side the Babinski sign was present, but delayed. The knee-jerks were exaggerated on both sides. Ankle clonus was not obtainable.

The gait was fairly good, though the left leg was dragged slightly. Spasticity was absent.

Notes made in the clinical record on February 10, 1906, state that the history as given above is confirmed. The only remaining sign of his paralysis seemed to be a lessened ability to wink the left eye and a shuffling gait.

The patient stated that after the stroke he laughed almost constantly and uproariously for two years, and that he still did so, but to a much less extent. When he laughs his whole musculature is involved, and tears gather in his eyes. He also seemed to have a coarse, rythmical, to-and-fro tremor of both upper and lower limbs, this being more marked on the left side. On using the finger to nose test the tremor was increased. His memory was good.

Examination in October, 1906, showed no paralysis of the left upper extremity, and some doubtful paresis of the left lower. Ataxia, however, was distinctly present in both left extremities. Sensation was unaffected. The deep and superficial reflexes were increased on the left side and prompt but not exaggerated on the right. Plantar irritation gave extension of the great toe on the left and plantar flexion on the right.

At this time, though not to the same extent as formerly, the patient showed a tendency to break into uncontrollable laughter on a slight or no provocation. He would be talking seriously, and suddenly in the midst of his conversation he would begin to laugh even uproariously. He said he had had this tendency to uncontrollable laughter since his attack in 1889, and that he had never had it before. He said he sometimes made up his mind not to laugh, but it was unavoidable after he had conversed with a person for any length of time.

His laughter was of an explosive and spasmodic character, occurring only at intervals, some of these being long. It was not alternated or intermingled with spells of weeping or howling.

The lesion in this case was probably similar in location to that of the following case in which a necropsy was obtained. In both cases the lesions were right-sided.

For the notes of the following cases I am indebted to Dr. T. H. Weisenburg.

Case II.—*Apoplectic stroke with left hemiplegia and paralysis of the right third nerve; irrepressible weeping and laughing, chiefly the former; necropsy showing destruction of the right nucleus ruber and partial destruction of the peduncles, cerebral and cerebellar.*

G., a woman, aged fifty-one years. The patient's grandfather and uncle died of paralysis. Her personal history up to the present illness showed

normal health. On April 22, 1910, while in the bath-tub, she suddenly fainted and was brought to the Medico-Chirurgical Hospital within forty-eight hours in a semiconscious condition. She was irrational, that is, talked in an incoherent manner; her speech was thick and could not be understood, but she had a tendency to repeat words. At this time the patient had a flaccid paralysis of the left side of the body. The reflexes on the right side were normal. On the left the knee-jerk was absent, but there was present a Babinski reflex. Owing to the patient's mental state the condition as to sensation could not be determined. Ptosis of the right upper lid was present. In smiling only the right side of the face was moved.

In a short time the flaccid condition was succeeded by spasticity, with increase of reflexes, ankle clonus, and Babinski. On the right there was present a complete oculomotor palsy.

From the beginning of her illness the patient was very emotional, crying easily and then again laughing, but crying most often. Occasionally, especially in crying and sometimes in laughing, she would shout loudly. In fact, whenever anyone talked to her she would cry or laugh. Her articulation at first was indistinct, and it was with difficulty that she could be understood; but after a time this was not so marked, and toward the end of her life she could be understood without difficulty. There was never any disturbance in swallowing and chewing.

The patient died four months after the onset of the attack.

Gross examination showed an area of softening in the cerebral peduncle on the right side, destroying entirely the red nucleus, extending into the middle and inner parts of the foot of the peduncle and destroying entirely the intramedullary fibers of the right third nerve.

Microscopically the area of softening was limited to the right nucleus ruber, and the middle portion of the foot of the peduncle (the outer and inner portions being normal), and the intramedullary fibers of the third. The cells of the nuclei in the right third nerve were diminished in number but not diseased. The whole nucleus ruber on the right side was destroyed, the softening extending a little to the left of the median line in a cribriform manner, but not involving the nucleus ruber on the left side. The lesion extended downward into the uppermost part of the pons, its limit being the decussation of the superior cerebellar peduncles. Upward it was limited to the cerebral peduncle alone and did not extend into the thalamus.

Case III.—*Extensive disease of the right midfrontal and prefrontal region, causing a smiling or laughing aura.*

The following case is worthy of report because of its general clinical and pathological interest, and also in connection with the present contribution

because of the peculiar initial bilateral facial spasm or aura which was distinctly of the form of a smile or laugh. The lesions were chiefly in the emotive zone as described in the paper. The case would seem to show at least that the right midfrontal region, and this and a part of the prefrontal cerebral zones are the seat of centres which control the bilateral facial movements of emotional expression. It is to be noted that while most of the time the initial spasm was bilateral and assumed the appearance of a smile or a laugh, at other times it was a unilateral facial movement, this appearing to show that in the diseased area were separate centres for the control of unilateral and bilateral movements of the face.

W. R., aged twenty-nine years, farmer, was first seen in consultation with Dr. M. B. Oberholtzer. After preliminary examination he was admitted to the nervous ward of the hospital of the University of Pennsylvania, where examinations were completed, and the patient was then referred to the surgical service of Dr. Charles H. Frazier for operation. The report of the case as given below has been summarized, omitting some unimportant and negative details of the elaborate examinations which were made.

When an infant the patient had had spasms for a year or two, these ceasing and returning again at intervals of about five years.

Some years ago, probably ten, he began to have occasional peculiar seizures, not very severe in type. In these seizures he had what the doctor, who has seen it more than once, spoke of as a peculiar smile or characteristic smile. It was a smiling expression with a somewhat dazed look, the first appearance being quickly followed by slight or moderate twitchings of the left side of the face. This twitching would often come on as much as a minute after the beginning of the peculiar smile. For several years he had only this sort of spells, in which he did not seem entirely to lose consciousness, although he was somewhat dazed, his consciousness being changed or reduced.

Four years before coming under investigation he began to have much more serious spasmodic or convulsive attacks. These as described by his physician and members of his family were usually preceded by the patient sitting around looking queer, as if something was going to happen. He would then have his so-called characteristic smile, which in about one-half minute or more would be followed by twitching of the left side of the face. This facial spasm or twitching first affected the corner of the mouth, then the cheek in general, next the eyelids, and eventually the entire side of the face. Following this facial spasm, or sometimes during its course, the spasm would extend to the left arm, and in more severe seizures to both arms, but it always predominated on the left side.

Soon after the patient was examined at my office he went with a member of his family and Dr, Edward M. Williams to the University Hospital. While on the way to the hospital he had one of his incomplete seizures. Dr. Williams in reporting his observation of this said: "First was heard a peculiar cackling, high-pitched, explosive laugh. As soon as the face was in view the mouth was seen to be drawn up in both corners and the eyes had a somewhat dull or staring appearance. The patient continued walking during the attack but in a somewhat listless manner, taking short steps, and apparently not knowing where he was going. He was told to stand still until the attack was over. The seizure was not followed by any twitching of the face, arm, or leg. The laughing lasted about one-half minute, and during it there seemed to be some swinging of the arms, which may have been normal, but looked to be in excess of the usual swinging of the arms in walking. As it passed off the patient walked on as usual."

When a child, before he was seven or eight years old, the patient lost considerable weight, and continued to be lacking in this respect. During childhood he had measles, chicken-pox, and at the age of twenty-seven whooping cough. These illnesses did not seem to have any effect upon the spasms.

The patient's head was notably large and unsymmetrical, the right side being considerably larger than the left, especially the frontotemporal region. Inquiry elicited the fact that when the patient was eighteen months old he fell and struck the right side of his head, and after the fall this side became and continued to be more prominent than the left. The patient said that at the age of thirteen, while skating, he fell and again struck the right side of his head, and after this the enlarged side became still larger.

The habits of the patient were good; he did not use tobacco, and alcohol only in very moderate amounts. He denied all venereal disease, and both Wassermann and Noguchi tests made after his admission to the hospital were entirely negative. The family history presented nothing of importance.

The patient was a well-developed man, aged twenty-nine years. He had a good bony framework and his musculature was in good condition. His head was large, of a hydrocephalic type, with a broad, rounded vertex. His hair was black and of the usual amount.

The patient wrinkled his forehead equally and well, and closed his eyelids tightly and equally on both sides. He pouched out his cheeks with somewhat increased expansion on the right side and drew up the left corner of the mouth better than on the right. The masseter muscles were contracted well, possibly a little more forcibly on the right side,

which might have been due to the unevenness of his teeth. The tongue protruded in a straight line, there being no tremor. It was free in all directions in its movements. The soft palate moved equally well on both sides.

An eye examination showed that the cornea was clear, conjunctivæ somewhat pale, eyeballs large but not protruding, and the eyebrows very prominent. The eyes moved freely in all directions and there was no disturbance of the visual fields. The pupils were dilated and equal, reacting promptly to light and accommodation. His sight, hearing, and smell were good.

Numerous examinations, general and neurological, showed nothing abnormal except as noted. His lungs, heart, stomach, and viscera generally were examined, and he was also thoroughly investigated for sensation, motility, reflexes, and everything else usually included in a neurological examination. The results were negative.

The following are descriptions of several attacks which the patient had after admission to the hospital:

Dr. Frank D. Reckord, resident physician, made the following note:

"While I was standing but a few feet from the patient he suddenly gave a queer cackle-like laugh. Both sides of the mouth were drawn up and the face wrinkled as if in the act of laughing. This lasted but a few seconds. He then turned his face to the right and buried it in the pillow. At the same time he flexed the right leg on the thigh as if restless. During this time his eyelids were about seven-eighths closed, but at no time did he close them tightly or was there any noticeable tremor. The nose did not quiver—and the attack did not seem to disturb his sleep. In a half minute he again turned his head to the left and kept on sleeping in that position. The attack seemed to be confined to his face."

Other seizures noted were as follows:

At 9 A.M. there was a slight convulsive attack of the muscles of the face and drawing of the mouth to the left. It lasted about a minute, and the patient complained that the jaw was stiff. He had a similar attack at 9 A.M. the next day, and a half hour later he had a laughing aura not followed by convulsion.

Operation was determined upon, and it was planned so as to uncover the midfrontal and prefrontal region, that is, those parts of the frontal lobe cephalad of the precentral convolution. It was decided upon with the hope of either finding a diseased mass which could be removed or treated, or failing this, of using cortical excision.

The skull was opened on the right side by Dr. C. H. Frazier so as to expose the brain anterior to the Rolandic fissure, including a portion of the first, second, and third frontal convolutions. The appearance of the

dura was abnormal, being somewhat cloudy and cystic looking. On cutting the dura there was a gush of fluid and an almost immediate collapse of the dura. The dura was seen to be thickened and adherent to the pia-arachnoid, and these in turn to the brain substance underneath. When the membranes were incised and lifted as far as possible out of the field of operation, a strange looking mass was revealed, consisting of fluid mingled with a fatty, gelatinoid, stringy, irregular mass of tissue, resembling in no way brain substance. The cyst, if it could be so called, was divided by strands or portions of this irregular mass so as to give the whole a somewhat multilocular appearance. The diseased mass, as above imperfectly described, included every portion of the exposed area. This exposure in accordance with the measurements and our observation extended from the anterior boundary of the precentral convolution almost to the anterior pole of the brain and from a superior level about an inch from the mesal line about the Sylvian fissure below.

Small specimens removed from the diseased mass were examined in the laboratory of surgical pathology and the following report was made:

The specimen consisted of two small sections of friable tissue, whitish in color, about 1 cm. in diameter.

On microscopic examination a diseased condition was seen indicating a pathological process of some sort which had resulted in atrophy and other destructive tissue changes, so that little if any normal structure was apparent. Here and there a few large cells could be seen in a partial state of preservation, and there were many clear spaces in the tissue, due evidently to some dilatation of the bloodvessels. There was no evidence of tumor formation or of any specific inflammation.

LANTERN SLIDE ILLUSTRATIONS

The following lantern slides were used in illustration of points brought out in the paper:

No. 1. (*a*) Normal appearance of the face in a case of paralysis of emotional expression on the right side; (*b*) voluntary movement of the face on the side of the paralysis of emotional expression.

No. 2. (*a*) Bilateral voluntary movement of the face in showing the teeth; (*b*) paralysis of emotional expression on the right side of the face shown in laughing.

No. 3. (*a*) Partial one-sided voluntary paralysis shown in bilateral movement of the face; (*b*) decided emotional paralysis as shown in laughing (Spiller).

No. 4. Head of snarling dog (Darwin after Wood).

No. 5. Face of dog dissected (Bell).

No. 6. (*a*) Face of horse; (*b*) face of horse dissected (Bell).

No. 7. Chimpanzee, "Peter," showing anxiety and uncertainty (Witmer).

No. 8. Muscles of the human face (Darwin after Bell and Henle).

No. 9. (*a*) Face in laughter; (*b*) muscles used in laughing (McClellan).

No. 10. (*a*) Face showing grief; (*b*) muscles used in expressing grief (McClellan).

No. 11. Scheme of the motor and motor-emotive zones.

No. 12. (*a*) Actor imitating crying; (*b*) imitating care (Borée.)

No. 13. (*a*) Actor imitating *ha*, *ha* laughing; (*b*) imitating forced, half-hearted laughing (Borée).

No. 14. (*a*) Child spontaneously weeping; (*b*) child spontaneously laughing (Darwin).

No. 15. Gwynplaine (from L'Homme qui Rit, 1869, illustrated by Vierge).

No. 16. Permanent grin, from bilateral brain lesion.

No. 17. (*a*) Normal appearance of face; (*b*) beginning to laugh; (*c*) laughing (Brissaud).

No. 18. (*a*) Usual appearance of face; (*b*) laughing; (*c*) weeping, in pseudobulbar paralysis.

No. 19. Violent weeping with lacrymation in cerebro-bulbo-cerebellar sclerosis (Weisenburg).

No. 20. Expression of anguish with howling, in bilateral brain lesion.

No. 21. (*a*) Expression of grief with forced attitude, standing; (*b*) painful expression, forced attitude, sitting.

No. 22. French Assembly taking the oath of allegiance to the new Republic (McClellan after David).

No. 23. Cartoon of nude figures illustrating muscular action in David's picture (McClellan after David).

No. 24. Bilateral lesions of the lenticula and internal capsule (Lloyd).

No. 25. Lesions of the pons.

No. 26. Lesion of the dentate nucleus, cerebellar cortex, and superior cerebellar peduncle.

No. 27. (*a*) Scheme of the mechanism of emotional expression, after Obersteiner; (*b*) main subdivisions of internal capsule (Obersteiner).

No. 28. Sensory or afferent emotional mechanism.

No. 29. Motor or efferent emotional mechanism.

No. 30. New scheme of efferent and re-enforcing mechanism.

Nos. 31, 32, and 33. Slide showing eyes interchanged in faces of man and woman (Brunton).

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DISCUSSION

Dr. George McClellan: Who of us all can speak so authoritatively on such a subtle subject as Dr. Mills?

As for venturing to question his conclusions I will not be so bold, especially in the companionship of those whom he has chosen to speak with me on *the mechanism of emotional expression.*

It would be difficult, indeed, to add to the interest of Dr. Mills' lecture, but in the brief time allotted to me I will try to accentuate certain points to which I have given special attention; after first referring to one of the convictions of a man of great ability with whose writings you are all more or less acquainted. There are few here to-night who do not know Sir Lauder Brunton. I was surprised, one of the last times I saw him in his own house, when he referred to his belief that very little, if any, expression could be attributed to *the eyes themselves*, and that all seeming variety of emotion betrayed by them was due to their more or less immediate surroundings.

In illustration, he presented me with a couple of slides, which he had been in the habit of using at the time he taught the anatomy of expression

in his lectures at Edinburgh. They are ingeniously conceived. On one plate a pair of eyes, pure and simple, entirely without eyelids, lashes, or facial surroundings; on another glass slide are the face of a drunkard and that of a nun, the latter with brow uplifted as in prayer. The eyes of each face are omitted, but so arranged that they can be superimposed upon the first plate and the eyeballs seen in these varying settings, which certainly alter their whole effect and import to a startling degree. (I will show these to you presently). While admitting the value of his observations, I cannot entirely indorse the conclusion of Dr. Brunton.

It is certainly evident that the human countenance betrays changes of expression more through the surrounding eye accessories, the brows, the lids, etc., than by the eyeballs themselves, yet there are times when in the recognition of the emotions especially one is convinced that the eyes are the "*windows of the soul.*"

It is the muscles about the eyes, under different influences, which convey the typical expressions in *laughter*, *astonishment*, *horror*, *anger*, *disgust*, or *grief;* but it is not easy to believe that the distinction of these expressions could be conveyed by a series of glass eyes, however artfully managed or surrounded. Surely the light of merriment, the profundity of grief, the glare of hatred or of horror, are not mere optical illusions of our imperfect vision, any more than the sudden flash of surprise or delight could be stimulated by a doll or a puppet with the cleverest mechanism.

How without the eyes can be interpreted the strength, the abandonment, or the intensity of these expressions, which are all produced by the same facial muscles with varying contractions? Surely each would lose all vital force and meaning if the eyes themselves did not play a leading part in them. The artist tries to illustrate the vital spark when he indicates the effect of the light upon the conjunctiva. Photography sometimes reproduces the high lights, but too often with a set expression in the muscles about the eye. (I will show you some slides of my drawings taken from my book *Anatomy in its Relation to Art*, which were suggested by a remarkable work entitled *Symphonies d'Expression*, by Maurice Heyman.)

The subtle distinction made by Dr. Mills between histrionic expression, whether caused by simulated or genuine emotion, and the undoubted effect of spontaneous feeling, in the expressions of the human face leads me to explain that these studies of emotional display, by Heyman, belong more properly to the former class.

As is well known (to anatomists) the muscles of expression are all of finer texture than the muscles generally in the other parts of the body. These muscles have no proper tendons of origin or insertion, are destitute of sheaths and blend with adjacent muscles by the most complex and delicate interlacement of their fibers, so that they do not present the same

arrangement in every individual. They even vary in development on the sides of the same face, sometimes thereby influencing the character or degree of the expression. (This is curiously conspicuous in the statue of Voltaire at the Theatre Français, Paris, and of Blackstone in the Bodlein Library at Oxford.)

In seventeen consecutive dissections of the face I have found no two faces with equal development of the muscles on both sides. The chief deviation had been in the zygomatic muscles and those about the angles of the mouth. The intimate association of the skin with many of the adjacent muscles produces through their habitual use the facial dimples, furrows, and markings, characteristic of certain expressions. This is especially noticeable in young children.

In the ignorant who cannot readily communicate ideas or describe sensations by speech, these lines are most valuable as indications, but they are usually very much exaggerated. It should be remembered that the changes of the features in the human countenance, where they are not due to the influence of the mind, but are expressions characteristic of strong emotions, passions, and affections, are the physical manifestations of impressions upon *the heart and lungs*. So are also the contortions of a child's face at birth, in this case almost automatic, before the brain is capable of mental effort.

The habitual expressions which stamp the features with individual character are often due to the exercise of the will in self-control, or to brooding of the mind upon certain passions.

Reverting to the eyes and the muscles which act upon them, elevation of character is often indicated by lines upon the forehead, which result from the habitual play of the muscles surrounding the eye and the muscles of its orbit.

Sir Charles Bell attributes to the eye the loftier emotions and describes the effect of its influence in reverence, in devotion, in agony of mind, in all sentiments of pity. The effect of power is greatly due to the projecting eyebrow. Beauty usually implies a large eye, and it is in the eye that one looks for sentiment and reproof.

The straight muscles of the eyeball are governed by the will alone, but the oblique muscles have sympathetic filaments which bring them under the influence of the sympathetic nervous system, so that they act when the other muscles cease to operate. Hence in sleep, in fainting, or in approaching death the eyeballs are revolved. In forced inspiration we instinctively droop the lids, and in sneezing and coughing it is the same.

The group of muscles of the upper part of the human face which by their action produce the so-called *intellectual expressions* give character to the features according to the predominant traits of the individual, even in repose, or after death. Bell pointed out that their function is to pro-

duce the *nobler* expressions of the human countenance, in contrast to that of the muscles peculiarly *animal* which act upon the lower part of the face.

Careful study of the lines of the face and their varying modifications prove of great value to the physician in forming a diagnosis of many diseases, especially in young children, and the ignorant, in whom the facial expression is often the sole guide to the recognition of subjective symptoms. The only way of obtaining information from an infant, who cannot communicate ideas or describe sensations by speech, is by noticing the expressions and gestures.

It should be noted that the expression of the emotions is not confined alone to the countenance, but that coincidentally there are movements of the rest of the body with characteristic postures and contortions, which participate in conveying the meaning. This is particularly the case in the self-abandonment of the savage among all races; yet it is a curious contradiction in fact, that while self-control is undoubtedly one of the acquirements of civilization, no statesman nor diplomatist can boast a more absolutely non-committal countenance and demeanor than is presented by the North American Indian, when such is his will.

That wonderful nerve, the vagus, is chiefly concerned in expression, through its connection with the larynx, the heart, the lungs, and the stomach. Of course, too, the facial and auditory nerves play conspicuous parts, and it is to be emphasized that the origins of all these nerves are very close to one another—mostly in the floor of the fourth ventricle (of the cerebellum), and it is not difficult to conjecture that nerves of communication may be established with certain areas of the cerebral cortex, especially with the frontal and central convolutions, and the cuneate lobes, as Dr. Mills has so clearly and ably pointed out.

Dr. James Hendrie Lloyd: The subject is widely speculative, but I think it is a great deal better that we confine ourselves to what we have learned at the bedside and the autopsy table. Dr. Mills has given us some illustrations of automatic or bulbar laughter, one of which was taken from a case of my own. We have in these cases a paralysis of certain muscles of the face, throat, neck, tongue, and sometimes of the eyes, caused by lesions above the nuclei of the third, fifth, seventh, tenth, and twelfth nerves. The lesions are frequently in the lenticular nucleus, cutting off the nuclei of these particular nerves in the midbrain, pons, and medulla from the cortical centres in the brain. It is in these cases particularly, called pseudobulbar palsy, that we have automatic laughter, in which the patient is seized with paroxysms of uncontrollable laughter or sometimes of weeping. It is a misnomer to call it, as some have done, epileptic laughter, for there is nothing epileptic about it. Dr. Mills has exhibited illustrations of the microscopic sections in my own case. These

accord with some few of the other cases reported in the literature. The lesions are in the lenticular nuclei on both sides and must cut off the corticobulbar fibers, *i. e.*, the fibers running from the motor centres in the cortex of the brain to the nuclei of the cranial nerves in the pons and medulla. There are a great many speculative theories about this phenomenon of automatic laughter. In the first place, if you come to think about it, this whole subject of laughter and weeping is curious. Why do we laugh or weep? There is no necessary or logical connection between the emotional condition in the mind and these peculiar spasmodic motions of the face which we call laughter and weeping. This function is confined to the human race, for the lower animals neither laugh nor weep. Since the days when our ancestors were apes we have in some way by long practice and habit evolved this peculiar function called laughter and weeping. My own theory is not so elaborate as that of others. My explanation is that we have evolved a sort of coördinated automatic apparatus which goes off practically almost by itself. It is like other automatic or semi-automatic functions, such as walking, talking, and writing, and can best be illustrated by an example. As a student I remember that Professor Gurney Smith used to show us a decapitated frog. When there was put upon the frog's leg a little drop of acid the creature would try with the foot of the other leg to scrape it off. Something like it is seen also in the decapitated rattlesnake: if its tail is trodden on the creature rears its headless trunk and strikes. This looks like a volitional act, but it is purely automatic. I do not think it is necessary to make up an elaborate series of explanations and schemata, which, indeed, to me are rather confusing. I believe the simplest explanation is the best; and in these cases (just as in the decapitated frog) the lesions in the lenticular nuclei act to cut off the volitional centres in the brain from the mechanical centres in the bulb.

Dr. Smith Ely Jelliffe: It gives me great pleasure to be here and especially to have heard the delightful address that Dr. Mills has given us, to have seen the beautiful photographs and to have learned so much from those who have preceded me in the discussion.

It seems to me, for myself at least, there is very little to add to the discussion, and yet I cannot refrain from saying just a few words that have suggested themselves to me while listening to Dr. Mills and seeing Dr. Weisenburg's splendid cinematograph demonstrations. I have prepared no elaborate discussion, and only wish, as it were, to give my own psychosensory reflexes as a result of the stimuli that I have received in the preceding hour.

This is a very old subject. Homer devoted considerable space to the question of sadness and of joy. Pliny said that the centre for laughter was in the diaphragm. An early Latin poet said, "Beware of the laughter that

shows itself only in the mouth." So that it has been recognized from time immemorial that the motor manifestations underlying two very primary principles in life—pleasure and pain—so far as emotional expression is concerned, show very complicated mechanisms. The mechanisms of emotional expression, are not as simple as Dr. Lloyd would have us believe, nor do the demonstrations we have seen permit us to dispose of the matter by the simple scheme suggested by Dr. Lloyd. If man were a simple spinobulbar animal, then the elementary facts touched upon by Dr. Lloyd would be sufficient, but fortunately we are not frogs. Human evolution has permitted the development of various cortical and subcortical structures. In order to excite these new psycho-motor-sensory reflexes an extremely complex mechanism has developed.

In the first place, I have heard nothing of the influences of sensory stimuli coming through the trigeminus. This sensory distribution of the face gives us all our information about the way the muscles of our face are set. Its functional integrity determines the postural muscular adaptations as well as the efficacy of the willed movements and the psychoreflex movements. The trigeminus is only one of the important factors in the parallelogram of forces. We must think of the subject of emotional expression as a psycho-sensory-motor reflex. I believe we have a vast polygon of forces to be analyzed from the motor as well as from the motor side. Thus in pseudobulbar paralysis there is no emotion with the laughter which we see. Here there is a modified reflex action, not an emotional disturbance. The subjects do not feel happy. They do not feel sad.

I am in accord with Dr. Mills in assuming that Bechterew's view will not hold. The thalamus is an important station for the reception of practically all of those sensations which underlie our affective reactions. In this sense it is a centre, not for emotional expression, but for perceptions, its disorders influence emotional expression but they are not responsible for the impulsive laughter and crying mimicry movements. These we shall find in a number of places in the cortex, in the midbrain, in the pons, medulla, cranial nerves, and in the spinal cord. All contribute elements to that polygon of forces, the disturbances of which result in the modifications of emotional expression so well illustrated this evening.

Dr. William G. Spiller: I know of no paper in which this subject has been treated so comprehensively as in the paper Dr. Mills has presented this evening. I also know of no case in which the paralysis of emotional movement has been so distinct as in Dr. Mills' patient. I have been interested in observing that in a number of persons the movements of the face in emotional expression are deficient on one side. It is rare that this leads to any misconception as to paralysis, yet last year a patient was brought to me in whom facial paralysis was feared, because of imperfect

movement of one side of the face in smiling. I have never seen bilateral deficiency of emotional movement.

Probst discovered that after destruction of the cerebellum there is impairment of muscular sense and of the sense of position, from which the conclusion may be drawn that a part of the sensory conduction is through the cerebellum. The loss of emotional movement, which movement is largely a reflex, may depend on the loss of these sensory fibers passing through the cerebellum. They may have their course in the fifth or seventh nerve, and I believe they are represented in the latter. Head has shown that the deep sensation is dependent on the nerves to the muscles, and I believe such fibers probably exist in the seventh nerve. Sherrington has demonstrated that the ocular nerves contain afferent fibers, and the function of these must be for muscular sense. I doubt whether any nerve has purely efferent fibers.

Dr. Augustus A. Eshner: Naturally there are a number of phases to be taken into consideration in a discussion of emotional expression. Dr. Mills has dwelt most largely upon the muscular expression of this phenomenon. Naturally no little consideration must be given to the exciting factors, and in connection with these the individual susceptibility to such stimuli also must be taken into account. As is perfectly well known, of any group of individuals exposed to a given psychic stimulus there will be varying degrees of response in the way of emotional expression, whether of sadness or of joy respectively.

There is another point to which I shall allude and to which Dr. Jelliffe has called attention, namely that in cases of bulbar paralysis and multiple sclerosis the muscular expression of emotion is not necessarily indicative of any affective disturbance, but represents merely muscular acts in response to influences beyond our immediate observation, and they may be considered, it seems to me, as rather counterfeits of emotional expression.

So, too, with the histrionic reproductions of emotional states. These, I take it, are mere superficial imitations, and naturally cannot be considered as expressive of deep-seated emotions. I doubt that any individual, however clever his histrionic ability, is capable, beyond a certain limited degree, of reproducing a true emotion, except in this superficial form, which must in the final analysis be considered merely as a simulation or as a counterfeit.

Dr. J. Cardeen Cooper: I did not hear Dr. Mills' paper, but there are two points in the discussion to which I should like to refer. One was made by Dr. McClellan in saying that the blank and sullen expression of the face is only noticeable in man, and is the result of education and civilization; and yet he refers to the North American Indian as showing often a marked degree of this tranquillity. It was said of Napoleon I that he

had this ability of expression to a greater degree than any other man of his time. Carlisle in his *History of the French Revolution* said that this was not due to education, but rather to his supreme will; and I have read somewhere that this absence of emotional expression is true of the North American Indian. It would seem, therefore, that the person did not have to be educated in order to dismiss from his face all lines of facial emotion and expression.

I wish to call attention to one point made in Dr. Lloyd's discussion, "that man is the only animal that shows the emotion and expression of laughter or grief." Dr. Pleuvences, of Paris, has said that Darwin in his *Descent of Man* says that all animals, such as cattle, dogs, and horses, show the very same expressions of laughter, grief, and various emotions as does man, and that the difficulty is in the inability of the observer to detect these expressions in the particular facial display of the muscles. Attention has also been called to the fact that a retriever, invariably, when a bird has been wounded or killed and brought to its master, would show the most decided expression of laughter in his face. Dogs also show a most decided expression of grief when conditions pertaining to grief are present.

Dr. Mills, closing: The College has already been liberal in the time allowed for the paper and the discussion. I will say only a word in regard to a remark made by Dr. Jelliffe in reference to the part played by sensation. The subject is so large that I gave only the merest outlines of what I had to present. If I had had time to demonstrate the three schemes of the mechanism, of emotional expressions which were thrown on the screen, he would have seen that one was illustrative of the sensory or afferent side of this mechanism, and the importance accorded to sensory pathways and centres would have been more evident.

AN EPIDEMIC OF EPITHELIOMA (MOLLUSCUM) CONTAGIOSUM, WITH SOME NEW OBSERVATIONS CONCERNING THE "MOLLUSCUM BODIES"[1]

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According to the statistics of the American Dermatological Association, molluscum, or better epithelioma contagiosum, comprises a little more than one-sixth of 1 per cent. of all diseases of the skin observed in America, and only a slightly larger proportion—one-fifth of 1 per cent.—is seen, according to Crocker, in the hospitals of London. From 1896 to 1906 inclusive only 11 cases of this disease were treated in the Skin Dispensary of the University Hospital, and in four of these ten years not a single case was seen. In 1907 there were 7 cases, in 1908 there were 12, in 1909 there were 6, while in 1910 no less than 21 were recorded in the dispensary in the course of a few months. Practically all the cases seen in these four years were from a common source, viz., a large institution for young men. Owing to the sudden increase in the number of cases from this source, it was decided to examine the entire student body in order to learn just what proportion was affected, but after examining 345, it was found to be impracticable, for various reasons, to continue with the examination. Of the 345 examined, 17, or about 5 per cent., were found to have the disease; but owing to the hurried way in which it was necessary to make the examinations, chiefly on account of the very limited time at our disposal, a certain number of cases in which the lesions were small and few, doubtless escaped detection so that the proportion of cases

[1] Read April 3, 1912.

was probably somewhat larger than these figures would indicate. If the same proportion held good in those not examined, and it probably did, there must have been at this time no less than 250 to 300 cases in the institution. In addition to those treated at the dispensary a small number from the same source were seen in my office, and a considerably larger number were treated by other physicians in various parts of the city. From the foregoing it will be seen that the epidemic was very extensive, amounting to some hundreds of cases spread over a period of four years.

Apart from its extent and duration, this epidemic presented some interesting and unusual features. The eruption was practically confined to the covered parts of the body; in not a single instance, as I recall, were there any of the tumors upon the hands or face; they were upon the trunk, especially the posterior surface, and upon the arms, the lower extremities escaping entirely. The number of lesions present was in most cases large, amounting to scores, while in a considerable number there were hundreds of them. In a few instances the little tumors were arranged in a line, the inoculation evidently having taken place in a scratch. They were for the most part unusually small, the largest being no larger than a split-pea while the greater number were the size of a pin-head, and instead of being flat, as is usually the case, they were quite acuminate. A large number of the patients complained much of itching, indeed, the eruption was commonly spoken of as an itch, and the skin often bore unmistakable evidences of this itching in the shape of many small excoriations and blood crusts where the tops of the lesions had been torn off by scratching. In a few cases the tumors were quite red and inflamed from the beginning, surrounded by a rather circumscribed inflammatory halo such as is often seen about the bites of insects, and the small central opening was so minute as to require the closest examination to be seen at all. These lesions resembled acne papules so closely that in one case at least in which they were of this inflammatory type upon the back the true nature of the disease was not at first recognized, having been mistaken upon a cursory examination for acne.

There is apparently but little doubt that the contagion was conveyed from one to another in this epidemic by the bath towels used in the gymnasium. Proceeding upon this theory every student found with the disease was excused from further attendance upon the gymnasium exercises until cured, and special care was taken that the gymnasium towels, instead of being laundered *en masse*, as it were, should be laundered in such a way as to insure that each towel should receive a thorough boiling. The result of these simple precautions seemed to amply confirm this theory of the manner of the transmission of the disease, for with their adoption there was an immediate falling off in the number of new cases; and during the past year I have seen but a single case in a student, and that was not a new one, but had existed for a year or more, its long duration being accounted for by the fact that, the number of lesions being small and unattended by any annoying symptoms, nothing had been done for it.

The agency of the bath in communicating the affection has been recognized by a number of observers, among them the late Dr. Radcliffe-Crocker and Sir Jonathan Hutchinson. The former has reported a number of such cases under his own observation, and the latter noted that all his male patients were in the habit of going frequently to Turkish baths, where, in his opinion, the gloves and towels were the medium of contagion. In one of Crocker's cases, a woman who was in the habit of taking a Turkish bath every day in her own home, the source of the infection was traced to her son who had contracted the disease from his schoolmates and had infected the bath. In Damascus the malady is called by an Arabic name which means "the itch of the bath" (Norman Walker).

In the treatment of the cases observed in this epidemic a number of local remedies were tried, among them a lotion of formalin, one or two drams to the pint of water. This lotion seemed to be effective in several cases, as the lesions disappeared to a considerable extent, but it commonly set up such a dermatitis that its use had to be suspended from time to time until this subsided.

Upon the whole, the most effective application was pure carbolic acid applied with a pointed match-stick or wooden tooth-pick by boring into the central opening of each tumor. A single application of this kind was usually sufficient, the tumor becoming inflamed and soon disappearing.

In connection with the treatment of this malady it is of interest to refer to an observation made by Kistyakovski. This author found that a number of molluscum tumors present on a young man with syphilis became inflamed after intramuscular injections of mercury given for the syphilis and then diappeared. In view of the well-known fact, however, that inflammation with disappearance of the tumors sometimes takes place spontaneously, further observations are necessary to prove the curative influence of mercury thus employed. Sakurane has quite recently reported five cases cured by the internal administration of a decoction made with the grain of *Coix Lacryma*, a remedy long employed in Japan in the treatment of molluscum. In four of these five cases the lesions disappeared in from one to two weeks. In four other cases in which the same treatment was employed, but which were not included in the report because they could not be followed up by the author, the disease was said to have likewise disappeared.

The appearance of the tumors is usually so characteristic that their nature is readily recognized, but occasionally they present such variations from the usual type, both as to size and appearance, as to lead to error in diagnosis. Pringle observed a case in which the lesions, situated upon the scalp, resembled rodent ulcer; and some years ago I had a case under my own care in which a single lesion on the forehead at the margin of the hair was mistaken for epithelioma of the ordinary type and excised; subsequent examination of sections made from it revealed the error in diagnosis. Although usually insignificant in size they may in rare cases reach very large dimensions. Walter Smith has reported a case in which there were many tumors, one of which was three inches in diameter; and Laache has described one in which the tumor, situated upon the occiput of a woman, aged fifty-six years, was the

size of two fists. When the disease attacks the mucous membranes, as it may do, although rarely, the chances of error in diagnosis are greatly increased. Abrahams, at a meeting of the Dermatological Society of London, described a case in which, in addition to many lesions upon the skin resembling lichen planus, the tongue was the seat of large white patches resembling leukoplakia which were shown to contain molluscum bodies.

The mode of transmission, although probably usually indirect, through the intermediation of baths, towels, etc., is in some instances due to direct contact, as when the mother's breast is inoculated by contact with lesions on the cheek of her nursing infant. In view of the difficulty which always tends the attempt to inoculate the disease experimentally this mode of transmission must be rather infrequent; but a most remarkable example of what was probably direct inoculation has recently been reported in the Westminster Hospital Reports for 1909. In seven cases under the care of a particular member of the staff of the hospital infection of operation wounds with molluscum occurred, the operator being found subsequently to have molluscum tumors on the hand. In all of these cases the tumors were situated in the scar or its vicinity, and had appeared in from six to nine months after operation.

In the vast majority of cases the disease is, without doubt, acquired through contact, immediate or mediate, with another individual, *i. e.*, it is of human origin, but there is reliable evidence to prove that it is occasionally of animal, and more particularly, of avian origin. Its occurrence in the domestic fowls, such as the chicken, turkey, goose, and in pigeons and other birds, is well established; and a few apparently well-authenticated examples of transmission from these to human beings are to be found in literature. Salzer has reported the case of a young woman with molluscum of the lids which he believed was acquired through feeding pigeons affected by the malady which existed as an epidemic among them and proved fatal to many of the birds. Sir Jonathan Hutchinson has reported a case, also occurring in a young woman, in which the affection was traced to a pet dog.

Heller, commenting upon this case, thinks it should be accepted with some reserve, since, according to him, molluscum contagiosum does not occur in mammals; but the nature of the tumors on the dog seems to have been well established by microscopic examination. Constantinides has described an affection similar to molluscum contagiosum as occurring in horses in Greece, the tumors having an anatomical structure similar to that of human molluscum. Jürgens, in performing some inoculation experiments with avian molluscum, infected himself, getting a tumor on the thumb. Czoker succeeded in inoculating the comb of a fowl with human molluscum, but Török and Tommasoli failed in their attempts to transmit it to rabbits and hens.

Although the infection is usually a trivial one in the human being it has a very extensive literature, attracting attention largely because it is one of the few examples if not the only one, of an epithelial neoplasm definitely known to be the result of an infection, although the actual demonstration of the infecting agent has thus far not been accomplished. The nature of the peculiar cells found in the interior of the tumors, the so-called molluscum bodies, has long been the subject of lively discussion. At first they were regarded as parasitic organisms, and some observers, the chief of whom is Neisser, still so consider them, but most investigators regard them as epithelial cells derived from the rete mucosum which have undergone some form of degeneration. According to Unna they are only hyaline or colloid prickle cells, while C. J. White, who has quite recently studied the histopathology of the malady in a most painstaking and thorough manner, thinks that the change in the cells "is not a colloid or hyaline degeneration, but rather an extraordinary metamorphosis of rete cells into keratin;" and this author also expresses the belief that no one has yet succeeded in demonstrating any parasite in the tumor.

During the course of this epidemic four tumors were removed from as many different subjects for microscopic study, three of them being fixed and stained in the usual way, while the fourth, an unusually small lesion just barely visible, was fixed and stained

according to the method of Levaditi. The study of the first three gave results which differed in no essential particular from those obtained and described many times by other observers, but the fourth, which was found to be situated in a lanugo follicle, gave histological pictures which, besides exceeding in clearness and sharpness anything which I have hitherto seen, revealed some new and interesting features which I shall describe with some detail.

In the area occupied by the molluscum bodies it was easy to distinguish three quite distinct varieties of cell. First, and most numerous, occupying the central portion of the tumor, were many large, round, and irregularly rounded bodies with double contoured walls and segmented contents, the walls and the lines separating the segments being stained a deep brown and presenting on careful focusing under a high power a finely beaded appearance. In most of these cells the nucleus was still preserved, although usually much distorted, and was always situated eccentrically; in many of the cells it was flattened out into a thin concave disk closely applied to the inner cell wall, as if it had been forcibly driven against the wall by some force within the cell. The finely beaded appearance of the cell wall and of the lines dividing the cell body probably represented the remains of the so-called prickles seen in profile which are normally present in the cells of the rete. Even the oldest of these cells, those occupying the upper part of the tumor nearest the central opening, which in sections stained with the usual stains present a homogenous appearance, still showed distinct segmentation. The second form of cell far less numerous than the first, more deeply situated, and often lying in the midst of apparently normal epithelium, was oval in shape, had a thick laminated wall and contained an oval nucleus always situated at one pole of the cell where there was always more or less of a cavity, the remainder of the cell being filled with a felt-like mass composed of very short and fine fibrils. In a few sections some of these oval cells were found lying partly within another cell of the first form, an observation which, so far as I know, is new. The third and smallest variety of cell, of which only a few examples were seen, was a perfectly

oval body with a distinct double wall, entirely filled with a mass of fine fibrils in which no nucleus could be distinguished; but all the cells of this variety were so deeply stained that it was extremely difficult to make out details satisfactorily. This apparently represents a form of cell which has not previously been described in this disease.

In conclusion, it may be regarded as quite certain that the large cells first described are epithelial cells which have undergone some form of degeneration peculiar to this disease, but I do not regard the nature of this degeneration as at all definitely determined. The other forms of cell which I have described are probably also degenerated rete cells, but the evidence for this seems to me much less convincing than in the case of the first variety of cell, and, therefore, other possibilities are by no means excluded.

DISCUSSION

Dr. F. C. Knowles: I had an opportunity of seeing the cases described by Dr. Hartzell, and they were of extreme interest for several reasons: because of the large number of lesions present, the distribution being limited to the trunk, instead of, as in the great majority of our cases, in the neighborhood of the eyelids, the fact that the tumors were unusually small and mostly acuminated, and because of the intense itching. I have seen one or two cases in which the scalp has been involved and in a few others the penis and the scrotum exhibited the attack, but with the exception of the present epidemic and the few cases mentioned, the face is almost invariably the site of predilection. The contagiousness of this affection has been discussed over a great many years, and although the majority of investigators now believe the exanthem is contagious, the attack usually develops a considerable period after exposure to the affection. Six months to a year being the average period required. Pick, of Prague, has been the most successful experimenter in producing the disease by experimental inoculation. In a home with which I was connected, I saw and reported 59 cases of this affection in about 400 children aged under six years; in one instance I was able to produce a typical molluscum tumor after an experimental inoculation.

PARTIAL GASTRECTOMY IN A CASE OF MULTIPLE CARCINOMA OF THE STOMACH[1]

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Instances of multiple primary malignant growths are sufficiently rare to warrant their publication. The following case of multiple primary carcinoma of the stomach is not altogether unique, but the recording of similar conditions has been infrequent.

H. K., aged forty-eight years, was sent into my service at the Jefferson Hospital by Dr. O. L. Wingate, January 4, 1912, and gave the following history: His family history was absolutely negative, his previous personal history was also negative; he had used beer, coffee, tea, and tobacco in moderation; he had always enjoyed good health and considered himself perfectly well until June, 1911, when he first developed gastric symptoms. At that time he was suddenly seized with a vomiting attack on the street, and says that the vomited material was black, and that he thought it contained blood. Repeated similar attacks occurred through the summer, and he rapidly lost weight and color. In the fall, under treatment, he improved, vomiting ceased, and he regained some weight and color. During the two months previous to admission the vomiting returned, and there was again rapid loss of weight and color. In the four weeks prior to admission he had lost twelve pounds in weight. The interesting point in his history, in view of the subsequent findings, is that he never had any pain or symptoms of indigestion, and the slight discomfort which he felt showed no

[1] Read April 3, 1912.

regularity as to the time of its occurrence. His chief complaint was vomiting, which did not occur every day, but he often vomited in the evening the food which he ate at breakfast. He thought that he had vomited practically all of the food taken during the few days previous to admission. He also complained of weakness and of tiring easily.

Examination showed a thin, very pale individual of distinctly cachectic appearance. Palpation of the abdomen revealed no tenderness, rigidity, or tumor. The blood examination showed: Hemoglobin, 44 per cent.; red cells, 3,140,000; leukocytes, 12,000; color index, 0.7. The gastric analysis was: Free hydrochloric acid absent; total acids, 0.0584; combined acids, 0.02185; no lactic acid; no bile; considerable blood; a few Oppler-Boas bacilli; a few yeasts; a few epithelial cells. A diagnosis of probable gastric cancer was made, and the patient was sent to Dr. Manges for *x*-ray examination. The plates showed marked irregularity in the outline at about the middle of the greater curvature and again near the pylorus in the greater curvature. The motility of the stomach was considerably interfered with, but Dr. Manges did not think that there was an obstruction of the pylorus; his interpretation of the plates was that the condition was malignant and that there were two distinct lesions.

Operation. On January 27, 1912, under chloride of ethyl-ether anesthesia, preceded by morphine and atropine, the abdomen was opened through the left rectus, and at once a large round mass at about the middle of the greater curvature was found involving both the anterior and posterior walls. The mass was densely hard, and there were small nodules beyond it; the gastrocolic omentum was puckered and attached to the growth. About an inch from the pylorus, on the greater curvature, was another hard mass, with an excavated centre, which could be easily felt through the anterior wall. The whole stomach was freely movable; there was no pyloric obstruction; no adhesion to the pancreas; no marked enlargement of the glands. The case seemed a good one for resection of the stomach, and I thought that there would be enough of the organ left for a gastrojejunostomy. The blood supply of the

area to be removed was first tied off, a ligature placed about the duodenum, and a clamp placed obliquely across the stomach from a point well above the larger growth to the cardiac end of the lesser curvature. The portion of the stomach included between the ligature and the clamp was then excised; the remaining pouch of the stomach was closed with two continuous catgut sutures and inverted with a continuous suture of linen thread. As the ligature around the duodenum did not completely close it, the cut end of this bowel was closed and inverted. I was surprised to find that the larger growth was adherent to the mesocolon, and I feared at first that the transverse colon itself was involved in the growth. I found, however, that I could trim away the adherent peritoneum without opening the bowel. The jejunum was then brought up through an opening in the mesocolon and attached to the posterior wall of the cardiac end of the stomach by catgut and linen sutures. The patient lost little blood during the operation, which occupied about an hour, and at its conclusion his pulse was 86.

The patient's convalescence was perfectly satisfactory excepting that he had a slight infection of his wound, which necessitated his remaining in bed longer than usual. He was discharged from the hospital on March 7, 1912. I saw him last on March 30, when he told me that he was able to eat all kinds of food and had no discomfort whatever. In the three weeks which had elapsed since his discharge he had gained twelve pounds. He was still pale, but his color was much better than before operation, and although I hesitated to prognosticate a cure, I felt confident further improvement would take place.

July 23, 1912. The patient died on June 19 of general debility, but without the recurrence of any gastric symptoms whatever. His only complaint was weakness in the legs, and it was thought for a time that he might have a spinal metastasis, but this could not be definitely determined. He did not die in the hospital, and no autopsy was made.

As the principal interest in this case centres in the character of the growths found, I am including a full pathological report from the laboratories of the Jefferson Hospital.

"Pathological Report, January 27, 1912. Patient H. K. Specimen, stomach. The specimen is an irregular portion of stomach wall, measuring 15 cm. in length and varying in width from 13 cm. at the broader to 7.5 cm. at the narrower end.

"The mucous surface bears two ulcers. The larger extends to within 1 cm. of the broader extremity. It is oval in outline, being 7.5 cm. long and 6 cm. wide. A peripheral zone 1.5 cm. wide is elevated from 0.5 to 1 cm. above the surface of the stomach. The surface of this raised margin is quite uneven, and has many reddish areas where the mucosa is lacking, the latter for a large part of the circumference forming only the outer wall of the elevated portion and not covering its surface.

"Internally the elevated zone dips down abruptly to the floor of the ulcer proper, which is 3 by 3.5 cm. The floor is fairly smooth and appears to be formed by the muscle coat, although this cannot be stated with certainty. Near one margin of the depression is an almost circular area 1 cm. in diameter that is much deeper than the remainder. The floor of this small area is formed by the inner surface of a grayish nodule 2 cm. in length and 1 cm. in height, that is attached to the peritoneal surface of the stomach; around the nodule is a narrow irregular zone of infiltration of the peritoneum that is but slightly elevated. This nodule is quite readily separated from a part of its attachment to the tissue surrounding the ulcer, this separation causing the appearance of an opening into the latter; apparently the escape of stomach content had been prevented only by this adherent mass, the stomach wall having a complete circular perforation 1 cm. in diameter.

"The smaller ulcer is toward the narrow end of the specimen and 1.5 cm. from the border of the large one, with which it has no demonstrable connection, either superficial or deep. It is 3.5 by 5 cm. in size, the peripheral portion being elevated as in the larger one. This elevated part varies from 0.8 to 1.5 cm. in width, and is covered by mucosa except for a few points along the narrower portion. The depressed portion or ulcer proper is 2 cm. in diameter, with a fairly smooth, grayish floor, probably formed by the muscle coat of the stomach. The peritoneal coat is slightly elevated and thickened over the area corresponding to this lesion

"The gastric mucosa not involved by the ulcers is rather smooth, grayish red in color, and is markedly corrugated, especially in the vicinity of the ulcerated areas. Incision of the raised margins of the ulcers encounters decided resistance, and the cut surfaces are somewhat granular, grayish yellow in color, and quite uniform in texture.

"Small portions of each mass were fixed in alcoholic corrosive sublimate, cleared in cedar oil, embedded in paraffin, sectioned, and the sections stained with hematoxylin, eosin, and Van Gieson's mixture.

FIG. 1.—Portion of stomach wall, showing the larger and smaller ulcers.

"Histology: Sections from the margin of the smaller of the two ulcers possess a partial border of gastric mucosa, which for the greater part shows but little change other than moderate infiltration by small round cells. In the depths this cellular infiltration is more prominent, and there is a moderate increase of intertubular connective tissue. Immediately below this is a rather abrupt transition to a newgrowth which has partly or entirely replaced

the muscularis mucosæ and forms the remainder of the sections. This new growth is made up of tubules, very irregular in size and shape, which are separated by bands of connective-tissue stroma that in areas is well marked, in others less prominent. The stroma is possibly residual submucosa (from its position), or is new tissue developed as a part of the growth. The tubules are lined by columnar epithelial cells, mostly in a single layer, but in some tubules stratified. These cells are placed directly upon the fibrous

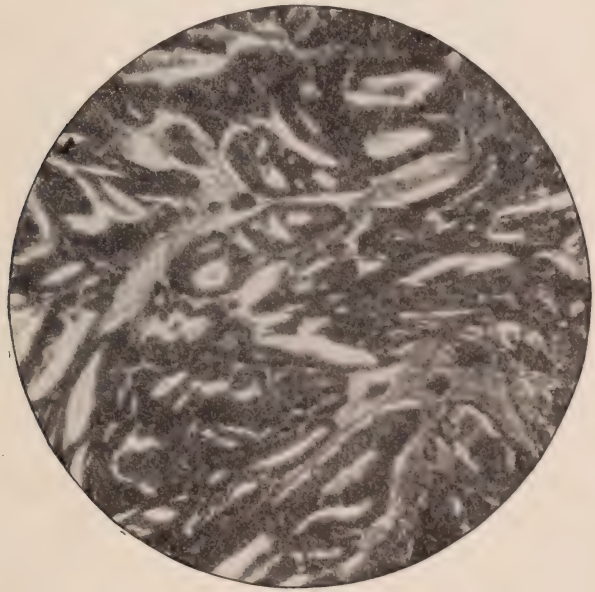

FIG. 2.—Photomicrograph of section of smaller growth.

stroma. Some of the tubules are partly filled by granular debris or desquamated and degenerating tumor cells. The stroma shows but little cellular infiltration.

"Sections from the wall of the larger ulcer also show a partial boundary of gastric mucosa. In areas the tubules at about their midpoint gradually merge into a structureless mass of rounded or polygonal cells which extend to, partly replace, and continue beyond the muscularis mucosæ as the remainder of the section.

There is in many areas no definite stroma, in this mass of cells forming the newgrowth, connective tissue being very scanty. At each boundary of this area of transition of mucosa into the tumor the latter extends below the muscularis without involving the overlying mucosa, the contrast between the structure of the two being very striking.

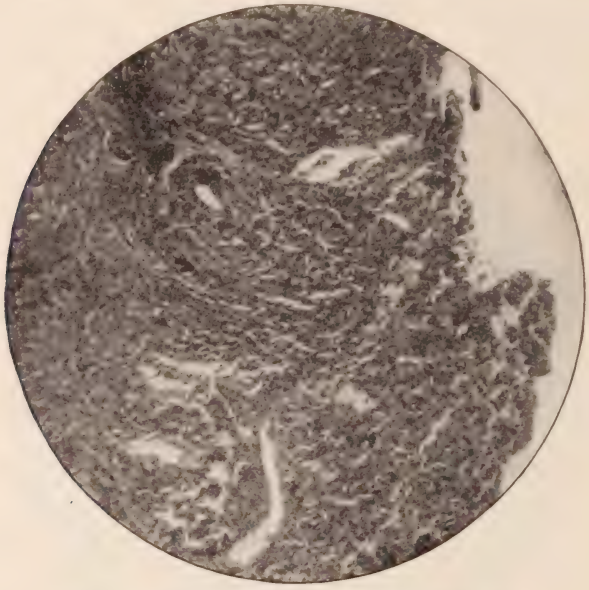

FIG. 3.—Photomicrograph of section of larger growth.

"Diagnosis: Both ulcers show carcinoma, which in each is in direct relation with the gastric mucosa. The structure of each, as above described, appears to justify the diagnosis of adenocarcinoma of the smaller ulcer and medullary carcinoma of the larger. In addition there is anatomical perforation of the larger ulcer, the opening being blocked by a mass of adherent newgrowth, presumably in a perigastric lymph node."

Dr. Despard and I have searched the literature dealing with multiple malignant growths, and one of the best accounts that we have been able to find is in Fenwick's[1] work.

[1] Cancer and Tumors of the Stomach.

Fenwick studied 1850 cases of cancer of the stomach, and in 54, or 2.9 per cent., multiple tumors were found, in three-fifths of these 54 cases, however, the tumors occupied corresponding spots on the anterior and posterior walls, and one showed a later development of the same type of cancer as the other. These were, in all likelihood, instances of transference by contact. Thus in about 1 per cent. there was no possibility of multiplication by contact, but in a number of these cases the growths were of exactly the same type and consequently can hardly be described as instances of multiple primary cancer of the stomach, a rare condition.

One of Fenwick's own cases presented a soft spheroidal-cell cancer of the cardia and a cylindrical-celled cancer of the pylorus. Several similar cases have also been collected from the literature by Fenwick.

D. D. Stewart,[1] reports a case operated upon by Dr. W. W. Keen, in which there were two isolated carcinomatous ulcers of the stomach: one was astride the lesser curvature of the stomach, and the other astride the greater curvature. The stomach in this case was removed post mortem, and a photograph of the specimen accompanies the report. Dr. A. O. J. Kelly examined the specimen and diagnosticated the condition as adenocarcinoma.

In Osler and McCrae's work on *Carcinoma of the Stomach* a case is reported of multiple submucous carcinoma of the stomach found at autopsy. The largest of the growths in this case was polypoid in character. The other growths were similar but smaller. From the histological character of the different growths Dr. Welch regarded the case as an instance of multiple primary carcinoma of the stomach.

Instances of multiple primary carcinoma occurring in different organs is more frequent than the occurrence of the condition in the same organ. Warthin[2] reports 2 cases of multiple cancer found post mortem. In the first there was an adenocarcinoma of the ovary and a colloid carcinoma of the gall-bladder. In the second case there was an adenocarcinoma of the sigmoid and an adeno-

[1] Amer. Jour. Med. Sci., November, 1898. [2] Jour. Amer. Med. Assoc., May 6, 1899.

carcinoma of the gall-bladder, each growth having started in a polyp and being of different anatomical character. In both of these cases stones were present in the gall-bladder.

Fenwick reports a case of spheroidal-celled carcinoma of the pylorus and a cylindrical-celled epithelioma of the rectum.

Albert Bauer, in an interesting inaugural address (Kiel, 1907) discusses at length the question of multiple primary carcinoma, and reports 3 personal cases. The first is that of "adenoma malignum" of the greater curvature of the stomach and "adenocarcinoma" of the pylorus, with glandular metastases from the latter. The second case showed an epithelioma of the esophagus, near the bifurcation of the trachea, and an adenocarcinoma of the stomach. The third case was one of squamous-celled epithelioma of the penis and adenocarcinoma of the stomach.

When two cancers of the same structure are found in the same organ the occurrence may usually be attributed to transference by contact or to autoinfection. It may be that the latter explanation would apply to my own case. The clinical history does not bear out the fact that we have a malignant change taking place simultaneously in two ulcers of the stomach. The patient was an intelligent man, and told me repeatedly that he never had any gastric symptoms until six or seven months before his operation.

DISCUSSION

Dr. W. M. L. Coplin: I had an opportunity to study slides from this case. An interesting factor from the pathological standpoint, is the question whether or not the cylindric cell or adenocarcinoma involving a glandular mucosa is or may be a preliminary or early stage of spheroidal or round-cell sarcinoma, so-called. A possibility that has not, so far as I know, been urged, is that in glandular mucosa possessing cylindric cells, the initial stage of cancer is cylindric cell in type, and that following the rapid proliferation of invading cylindrical epithelial cells a transformation gradually takes place, spheroidal cells eventually predominating. If, by any possibility, such a view be correct, then instead of

regarding these growths as essentially distinct primarily, it might be better to assume that one has either reached a later stage of invasion and evolution or is growing with such rapidity that the typical cylindrical cell is not produced and that the distinction is one of cell evolution rather than the presence of two essentially dissimilar types of growth. It is well known that in adenocarcinoma of the mammæ sometimes the secondary growths occurring in lymph nodes take on the attributes of scirrhous or encephaloid, and that very rarely squamous-celled epithelioma of the lip manifests something of the spheroidal cell type when invading the lymph nodes; it is true that such cases are exceedingly rare, but undoubtedly they occur. In other words, the matter finally terminates in a discussion of the unity of cancerous processes. Whether we have in all types of carcinoma a similarity of result from many forms of stimulation of epithelial growth which of itself does not determine the type of carcinoma or that the form is determined by the primary cause remains uncertain. The view that the histological type of a growth is determined by the morphology of the epithelial cell primarily involved may not be sustained in all cases.

Morphologically the two growths in this case are clearly distinct, and I have not seen in the sections evidence of intermediate stages. When looking at the two growths one would not suspect that they were from the same organ, much less that they were closely approximated.

THE IMPORTANCE OF REMEMBERING THAT ALL PULMONARY PHYSICAL SIGNS ARE NOT THOSE OF TUBERCULOSIS[1]

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Although some members of the profession seem at times to think that the physical signs of early tuberculosis of the lungs have been recognized only within recent years, as a matter of fact, they were well studied and thoroughly understood long before the bacillus of Koch was discovered. Those who had not the aid of the microscope in determining the presence of the bacillus were forced to train their special senses to a point rarely reached by their successors, whose auditory nerves are lessened in their acuity by the use of the phonendoscopes, and whose tactile sense in estimating fremitus is often untrained by lack of constant use.

The recognition of the fact that pulmonary tuberculosis is curable in the great majority of cases, if its presence is determined early in the malady, has been impressed so strongly upon the mind of medical men, and the disastrous results of oversight are so often met with, that there is a tendency to regard all abnormal physical signs in the lungs as indicative of tuberculosis unless the symptoms are so indicative of other affections that tuberculosis is manifestly ruled out. In other words, the fact that physical signs are present in the lungs is considered fair evidence that tuberculosis is the cause, and so individuals are classed as tuberculous when not so diseased. I am well aware of the fact that countless instances can be brought forward in which unrecognized

[1] Read April 3, 1912.

lesions have produced disaster, and I am not seeking to make light of the importance of recognizing that the early diagnosis of tuberculosis is essential. My object is to point out that some cases are not tuberculous, although thought to be so, and to emphasize the fact that there are no physical signs to be found in the chest which cannot be produced by lesions which are not tuberculous in origin. This point can, I think, be put in a concrete form by the citation of several cases:

Case I.—A girl of eighteen years had a history of constant cough for six weeks, with practically no expectoration. Her evening temperature often rose to 100°, and she had little appetite. Her loss of flesh amounted to about eight pounds. At the right apex, above and below the clavicle, there was some impairment of resonance on percussion, some prolongation of expiration, no rales, but slight tubular breathing. She had a history of a sharp attack of influenza at the beginning of her illness, but no adequate examination of her chest had been made. An examination into the amount of food she had taken daily showed that owing to her anorexia she had not received more than half the calories needed to maintain her weight for the six weeks spoken of. Indeed, it represented less than 1200 calories a day on the average. The diagnosis was impairment of normal lung action, because of infiltration or congestion of the apex as the result of the influenzal infection, with partial starvation. She was sent to the mountains, ordered proper amounts of food and exercise, and returned in about three months, having gained fifteen pounds and with no physical signs. She is still in perfect health. It may be claimed that this is an instance of a cured tuberculosis, but I think not.

Case II.—A man of forty-nine contracted a cough from his adolescent son who in turn had been infected by an epidemic of pertussis at a boarding-school. The paroxysms were so severe that he had to stop on the street to support himself by nearby objects when seized, although he never whooped. After some three months he still had a spasmodic cough and had lost twenty pounds. At his left apex there was prolonged expiration, blowing breathing, and high-pitched resonance on percussion. The loss of weight was due to the exhaustion produced by his cough, the physical signs I believe to be due to a localized emphysema of the lung. He has since become perfectly well save for the persistence of the physical signs to a moderate degree.

Case III.—A physician engaged in sanatorium work presented himself with a history of marked loss of weight, slight febrile movement, malaise, some anemia, loss of appetite and impaired digestion. At the base of

his right upper lobe posteriorly he had dulness on percussion and bronchial breathing. His tonsils were enlarged and their follicles filled with foul secretion. There was slight enlargement of the cervical lymph nodes on the right side. His tonsillar trouble was attended to and successfully. He was told that he had a pulmonary lesion and had better stop work and betake himself to a better climate. He then volunteered the information that some years before he had had a pleuropneumonia of the right side of great severity. With the clearing up of his tonsillar infection and proper general treatment he speedily recovered perfect health and the diagnosis of pulmonary tuberculosis was revised so as to read tonsillar infection and an old lesion resulting from pleuropneumonia.

Case IV.—A man of about twenty-one lost fifteen pounds in three months. He had a rise of evening temperature of two degrees and very profuse mucopurulent expectoration, amounting to several spit cupfuls a day. At one apex there seemed to be slight impairment of resonance, and some rales were heard. On both sides of the vertebra there were many rales and whistling or tubular breathing at several points. The diagnosis of dangerously advanced tuberculous disease of the lung was made. I took the liberty of revising this to pneumococcic infection of the bronchial mucosa with possibly some bronchiectasis. The use of expectorants, an outdoor life, and good food resulted in no expectoration, little cough, no rales and a gain of fifteen pounds in three weeks with no febrile temperatures. No tubercle bacilli were in the sputum but multitudes of pneumococci, although there was no history of pneumonia.

Case V.—A woman, aged twenty-one years, after a severe tonsillar infection followed by infection of the cervical lymph nodes, developed six weeks later moderate continued fever lasting several weeks, marked loss of flesh, constant unproductive cough, loss of appetite and loss of weight. She complained of a sense of discomfort at the third interspace near the sternum, which was increased by coughing. At this point there was distinct blowing breathing and dulness on percussion. The cough became slightly productive, probably because it caused tracheal irritation, but the sputum was negative except for a few pus organisms. The right apex showed distinct impairment of expansion. The diagnosis of early tuberculosis was made, but suddenly revised, when, after a severe attack of cough, a teaspoonful of pure pus containing no tubercle bacilli was expelled. Recovery speedily followed. Diagnosis, infection, and suppuration of a bronchial lymph node rupturing into a bronchus.

Case VI.—A woman, aged thirty years, remarkably athletic and strongly built, lost strength, health, and appetite, and had an incessant cough which failed to be benefited by ordinary measures. A cold shower bath following exercise gave a good reaction, but caused such violent

cough that chloroform or amyl nitrite was used to control it. Examination of the chest showed on the right side of the sternum, for two inches vertically, distinct impairment of resonance from the second to the fourth interspace, with tubular breathing. The *x*-ray revealed a chain of enlarged bronchial lymph nodes on both sides. As the illness had followed an attack of influenza, she was treated by rest and iodide of iron, and then sent South for the winter, with perfect recovery as a result, but she and her family were in misery for weeks because of the diagnosis of pulmonary tuberculosis.

Case VII.—A young man in his twenty-first year, a student at Yale, where he had been very athletic during the late fall and early winter, suffered from pain in the stomach, loss of flesh, cough, and loss of vigor. He became pallid and suffered from dyspnea on exertion. When examined by me he was found to have at the right apex tubular breathing, great impairment of resonance and a few rales. The diagnosis made was tuberculosis, the more so as he had an evening temperature of 101.5°. He became rapidly worse and the area of consolidation of the lung rapidly increased. At the request of the family one of the older Fellows of this College saw him and made a diagnosis of epidemic influenza. Some time later he was seen by the late Professor Da Costa, whose name I mention because any opinion expressed by him is so well worthy of confidence. His diagnosis was scrofulus, probably because the cervical lymph nodes were swollen. The blood examination was negative. The spleen was not materially enlarged. The disease ran a very rapid course, ending in death from respiratory obstruction. Toward the end the diagnosis was evident and the autopsy confirmed it, namely, Hodgkin's disease, with the lesions chiefly in the bronchial lymph nodes.

Case VIII.—A woman, aged twenty-eight years, caught a severe cold, which lasted for several weeks, with persistent cough. An examination of her right apex showed impairment of expansion, impairment of resonance, some prolongation of expiration, and a few rales. The diagnosis was probable tubercular infection, but perfect recovery of health in two weeks and the discovery of the history of a pneumonia in 1908 resulted in a diagnosis of an acute cold with physical signs, due to an old catarrhal pneumonia. I can cite many similar instances.

Another word may be said as to the diagnostic insignificance of dulness and absence of vesicular breathing at the base of the lungs. Nearly all of these result in the diagnosis of "consolidation" or "pneumonia." In severe bronchitis, in whooping cough, and after operations in the upper abdominal segment the real state is often atelectasis.

Finally, a few words as to bronchial lymph nodes. It is remarkable that although these nodes are universally recognized in books on morbid anatomy as being often diseased, the practitioner almost never regards them as important facts in diagnosis, yet these are often the cause of marked physical signs. Sahli describes Neisser's method of their diagnosis by the distended finger cot on a bougie and the *x*-ray will often show them. Delafield and Prudden say:

"The tracheal and bronchial lymph nodes may be the seat of a variety of lesions which, owing to their situation, as well as for other reasons, are of considerable practical importance. They may be enlarged from hyperplasia in acute infectious diseases; by the development in them of tumors; in leukemia and with special frequency in tuberculosis. They may become pigmented from inhaled coal or other dust, and may atrophy or become fibrous or calcified. In cheesy degeneration following tuberculosis, or in the suppurative inflammation, perforation may take place into the air passages, the esophagus, the pulmonary bloodvessels, or aorta, or into the pericardial or pleural cavities; in this way hemorrhage or secondary inflammatory processes or gangrene may occur. Death may occur from pressure upon the trachea by tumors of the adjacent lymph nodes. Sudden death from asphyxia may result from perforation into the trachea. Pressure upon the pulmonary veins may lead to pulmonary edema. The bronchial lymph nodes are very important as distributing centres of infectious microörganisms, and particularly as points of lodgement of tubercle bacilli, which have been gathered from the pulmonary air spaces from the pharynx or elsewhere." They also give the following references to other literature.[1]

[1] Consult for summary of lesions of bronchial lymph nodes Hall, Philadelphia Medical Journal, December 1, 1900, bibl. See also concerning tuberculosis reference Northrup and Bovaird, p. 487, and Harbitz, Jour. Inf. Dis., 1905, vol. ii, p. 143. See also statistics of Hand, Phila. Path. Soc., 1903, vol. vi, p. 132. For an excellent resume concerning normal and pathological tracheal and bronchial lymph nodes see Marfan in Bouchard and Brissaud's Traite de Médecine," t. vii, p. 525; also Zuber in Grancher, Comby, and Marfan's "Traite des Malasies de l'Enfance," t. iv, p. 235. For a study of the relation of the bronchial lymph nodes to the lymph vessels of the thorax, see Welemincky, Berl. klin., Woch., 1905, p. 743. For a study of the relationship of the cervical and bronchial lymph nodes, see Beitzke, Virch. Arch., 1906, Bd. clxxxiv, 1.

Again in Osler's *Modern Medicine*, Warthin says:

"An acute simple inflammatory enlargement of the bronchial glands occurs in severe forms of bronchitis, pneumonia, and other acute inflammations of the lungs. It is very common in children in association with the acute infectious diseases, particularly when these are complicated with respiratory affections. It is highly probable that in the majority of cases of simple cervical adenitis there is at the same time more or less enlargement of the bronchial nodes. In the ordinary cases the symptoms due to the pressure of the enlarged glands cannot be separated from those of the primary condition; but when the enlargement is marked, severe pressure symptoms may be produced. Dysphagia, dyspnea, respiratory stridor, cyanosis, and the physical signs of bronchial stenosis constitute the clinical picture. Should suppuration occur, the abscess may rupture into the bronchi and sudden death ensue. Chronic inflammation of the bronchial nodes is present in chronic bronchitis, bronchiectasis, chronic pneumonia, etc."

Finally, in Spitzka's edition of Gray's *Anatomy* we find these words:

"In all town dwellers there are continually being swept into those nodes from the bronchi and alveoli, large quantities of the dust and black carbonaceous pigment that are so freely inhaled in cities. At first the nodes are moderately enlarged, firm, ink black, and gritty on section; later they enlarge still further, often becoming fibrous from the irritation set up by the minute foreign bodies with which they are crammed, and may break down into a soft slimy mass or may calcify. Not infrequently an enlarged tuberculous node perforates into a bronchus, discharging its contents into the tube. When this happens there is great danger of acute or pulmonary tuberculosis, the infecting node substance being rapidly spread throughout the bronchial system by the coughing its presence in the air passages excites."

French has recently pointed out that the lymph nodes immediately below the right bronchus are often infected, and Siennikow has written a valuable paper in which he shows that these nodes are very large ones. By its pressure on the bronchus

it causes blowing breathing, and by its irritation of the vagus or phrenic it causes cough. There can be no doubt that one of the chief causes of disease of this node is tuberculosis, and a valuable paper on this subject is published by Jordan in the *Practitioner* for February, 1912.

My point is not to deny that they are often tuberculous, but that they may be infected by other organisms than the tubercle bacillus, and second, that many physical signs thought to have their origin in the lung tissues are due to partial bronchial occlusion by pressure. An interesting illustration of the value of the *x*-rays in clearing up this question is shown by these plates, which Dr. Manges has kindly brought here.

If anyone thinks, after hearing this paper, that I minimize the frequency of tuberculosis, that I fail to appreciate the disastrous results that comes to the relatives when a long drawn out and hopeless fight ends in defeat, he fails to grasp my meaning.

My object is not only to be always on guard against the ever present possibility of pulmonary lesions, but also to emphasize the fact that every subacute or chronic pulmonary lesion presenting impaired resonance, prolonged expiration, and, for a short time, febrile movement is not tuberculosis.

A most valued assistant said to me, when I spoke of this paper: "You may be right, but you will do a great deal of harm by making men careless as to the possibility of tuberculosis in a given case." I think not, because if any state of the lung, save a healed lesion, presents these physical signs the patient should be warned, and should be treated by fresh air, good food, and all other measures to increase vital resistance, which measures are identical with those for tuberculosis. In many instances, untold mental misery for the patient and his family is engendered by the sentence of tuberculous infection, and his whole manner and place of living is altered. His career is not only changed in its course, but probably narrowed or arrested. He is afraid to plunge into the struggle for success because he is in dread of a relapse or return of his "old trouble." His seniors are afraid to put him in line for advancement lest their act prove a mistaken kindness and cause his breakdown,

his chance of getting life insurance is stopped, and even his marriage may be prevented by the blot on the history of his lungs. These are not imaginary theories. I can point out man after man, in and out of the profession, who have been so hampered. When tuberculosis has been present, nothing but thanks for his recovery can be thought of, but where it has not been present, a mistaken diagnosis has been a calamity.

The parallel between the diagnosis of syphilis and tuberculosis is exact. Syphilis is very prevalent, it is the most likely explanation of many symptoms and signs, its correct diagnosis is essential for the sake of the patient, his career and his relatives, but before morale is shaken by being told that he has syphilis, before his life is changed and he is perhaps intimidated so that he is tied forever to a fear that an old malady is constantly with him, ready to develop at any moment, all the other possibilities of his case should be excluded.

We must be careful that the predominance of tuberculosis does not cause us to commit the patient to the constant woe of worry and alarm, until we have excluded the conditions which produce physical signs in the chest similar to those of tuberculosis.

DISCUSSION

Dr. James Tyson: I think Dr. Hare's paper is very timely, indeed all such papers are timely. It happens to me every year, more or less, to meet with cases of this kind coming under the category of which Dr. Hare has described the symptoms, where the patient has been under the impression that there is tuberculosis and this happens not to be the case. There are several ways in which unfortunate consequences arise from such a misinterpretation. Dr. Hare has mentioned a number of them, but in addition to the fact of the untold misery and the other disadvantages which grow out of the supposition that a person has tuberculosis, we have the matter of statistics very seriously interfered with. In the second place, the curability of consumption is exaggerated by such interpretations as those which he has narrated to us. There is one point to which Dr. Hare did not allude for which I looked, and that is the

matter of the coincident presence of bacilli. Many of these doubtful cases are cleared up by examination for the bacilli. I am free to confess that such a train of symptoms if accompanied by bacilli in the sputum would cause me to make a diagnosis of tuberculosis, whereas without such a finding I should have been in doubt, or made the opposite diagnosis.

Dr. Hare: I did not deal with the matter of bacilli because I think we all recognize that if we wait until we have the presence of bacilli in the sputum, we often wait until the disease has advanced so far that we can do little for the patient. Of course, if bacilli are found, even if there are no physical signs, one has the right to say that the patient has tuberculosis. I think that the skilled physician must discover the disease before the microscope discovers it for him.

THE TREATMENT OF PERSISTENT PAIN OF ORGANIC ORIGIN IN THE LOWER PART OF THE BODY BY DIVISION OF THE ANTEROLATERAL COLUMN OF THE SPINAL CORD.[1]

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REMARKS BY DR. SPILLER

Occasionally the pain from tumor of the cauda equina is so intense that complete division of the spinal cord has been suggested for its relief, and it is only recently that I have seen a case in which this measure had been proposed. If division of the anterolateral columns alone will remove pain, it may be possible, if the other parts of the cord be left intact, to avoid paralysis of the lower limbs and of the bowels and bladder by this operation. It probably would produce ataxia. Such an operation may be considered only in extreme cases when life has become almost a burden because of severe suffering.

A. Schüller[2] has suggested that as a substitution operation partial division of the spinal cord might be employed instead of section of the posterior roots for spasticity and gastric crises. The posterior columns, he suggests, might be cut for spasticity either alone or with the direct cerebellar tracts; the anterolateral columns might be cut for gastric crises. He reported no cases in

[1] Read April 3, 1912.

[2] Wien. med. Woch., 1910, p. 2292.

which this operation was performed, and he advised it for these two conditions.

I would suggest that the operation of division of the antero-lateral columns is applicable for pain independent of gastric crises, and that it may be much more than an *Ersatzoperation* for division of the posterior roots. So far as I know the case reported in this paper is the only one in which this operation has been done.

On what is this operation for the relief of persistent pain based? Are fibers of pain confined to one system within the spinal cord, and if so, can pain sensations be conveyed by other systems when the fibers usually employed for this purpose are destroyed? These are important questions.

Head and Holmes,[1] in their recent Croonian lectures, refer to the work of Rivers and Head, in which it was shown that beneath the skin, independent of all "touch" and "pain spots," lies an afferent system capable of a wide range of functions. Pressure which in ordinary life would be called a touch can be appreciated and localized with considerable accuracy. Increase of pressure, especially on bones and tendons, will cause pain. In the peripheral mechanism, therefore, there are two independent mechanisms for the initiation of pain. Within the spinal cord, however, the conditions are different. All impulses capable of generating pain become grouped together in the same path and can be disturbed simultaneously by an appropriate lesion of the spinal cord. In the same way, sensibility to heat or cold may be lost independently of one another, showing that all the impulses on which they are based have been sorted out into two functional groups, each of which passes by a separate system in the spinal cord. It should be possible, therefore, to divide the tracts for the conduction of pain sensation with little damage to the rest of the cord, except that the fibers of temperature sensation probably would be implicated. It is questionable whether objective disturbance of sensation would be produced by such an operation, and, if it were, whether it would be persistent.

[1] Lancet, London January 6, 1912.

Fabritius[1] recently has stated that little attention has been paid in neurology to the late results of interruption of the temperature and pain tracts in the spinal cord. It is in general assumed that the disturbance in these sensations gradually disappears, but if one attempts to determine on what this opinion depends he meets with difficulty. Fabritius was not able to find any extensive investigation of this subject, and he attempts to fill the gap by collecting cases from the literature. The summary of his findings is that after stab wounds of the spinal cord no regularity exists in the restoration of pain and temperature sensations. In the majority of the cases (23 out of 35) temperature sensation seems to have been permanently lost. Pain sensation returned more frequently, but in diminished intensity, namely, in 18 out of 38 cases, but was permanently lost in 16 cases. It is questionable whether all the statements regarding sensation in these cases can be accepted as accurate.

Rothmann,[2] in a paper read at the recent meeting of the Gesellschaft deutscher Nervenärzte, gave as the results of his investigations that pain sensations are conducted essentially through the anterior part of the lateral column, and that the gray matter may be concerned in this function, but concerning the latter statement the report is not very definite.

The work of Petrén[3] and my own studies[4] of the location of pain fibers within the spinal cord lead to the conclusion that fibers for the conduction of pain are located in the anterolateral columns.

In order to make a test of this reasoning in a very severe case of tumor of the lower part of the cord, when pain was intolerable, I referred a patient to Dr. Edward Martin for division of the anterolateral column on each side. Dr. W. B. Cadwalader and Dr. J. E. Sweet later performed the same operation on dogs to further our knowledge in respect to the function of these columns.

[1] Monats. f. Psychiat. u. Neurol., January, 1912.
[2] Ibid., p. 81.
[3] Arch. f. Psychiat., xlvii, 495.
[4] Univ. Penna. Med. Bull., July and August, 1905.

A man, aged forty-seven years, was admitted to the Philadelphia General Hospital, March 8, 1909, in my service, and came later under the care of my colleagues on the staff. He had had pain for two months in the knees and ankles, and at the time of admission had pain about the pelvis. The pain increased, and by August, 1909, he complained greatly of it and had almost complete flaccid paralysis of the lower limbs, with loss of tendon reflexes and paralysis of bladder and rectum. He had anesthesia over the buttocks and external genitalia and down the back of the thighs. The lower limbs were atrophied and faradic contractility was diminished.

Diagnosis. In November, 1909, introduction of the finger into the rectum caused no contraction of the internal or external sphincter. Dr. W. P. Hearn operated and found a growth on the left side and lower part of the spinal cord about as large as a goose egg, the cord being implicated by the tumor. It was regarded as malignant and irremovable.

In January, 1910, the man came again into my service and notes taken by me at that time record that he had very slight movement at each hip and in the left knee, and no movement elsewhere in the lower limbs. These limbs were greatly wasted. Sensations of touch, pain, heat, and cold were lost in the back of each thigh and in all parts of the leg below the knees, although deep pin-pricks seemed to be felt in the left calf and left foot. The tendon reflexes were lost and the legs were contracted on the thighs and the thighs on the abdomen. Sensation in its various forms was preserved in the abdomen and front of the thighs. The man suffered greatly from pain in the lower limbs and required morphine every night for relief.

Operation. The division of each anterolateral column was performed by Dr. Martin, January 19, 1911. On January 22 there was great relief of pain in both lower limbs. The patient appeared very grateful for the relief from suffering and received only $\frac{1}{6}$ grain morphine on the day following the operation and a similar amount two days later, and this was given for the pain caused by the operation. The interne on the surgical service, who had the opportunity of observing the man constantly, believed that he was not suffering pain in the lower limbs. Sensation in its various forms objectively tested did not seem to be much impaired above the hip-joints. Pain was felt occasionally in the lower limbs during the three weeks following the operation, but the man was positive that it was less than before the operation.

Course. In March, 1912, when he was again under my care, he lay in bed with his lower limbs greatly contracted and atrophied. Objective sensation was lost in the front of the thighs, but pain and pin-prick sensations were keen over the lower abdomen. His countenance expressed

no pain, and he never complained unless he was asked concerning his condition, when he would reply that he sometimes had pain. As he is a foreigner and not very intelligent, his condition is pitiable, and it is hard to judge whether he really has any pain from the tumor. The operation seems to have been successful in the diminution of pain. I should be unwilling to form an incorrect judgment regarding this case. When one remembers, however, that the pain from a malignant tumor of the lower part of the cord usually is excruciating and continues as the tumor implicates additional posterior roots, I think it may be said that the peaceful expression of this man's face is evidence that he has been greatly relieved by the operation. He has certainly not been made any worse, as his condition before the operation was very grave.

REMARKS BY DR. EDWARD MARTIN

The problem presented by Dr. Spiller was the making of a transverse cut into the spinal cord, roughly 2 mm. in length, of a similar depth and with its posterior end 3 mm. anterior to the entrance of the posterior root, this cut to be bilateral. The only special instrument required was a small thin-bladed cataract knife, double-edged on its angled point, the latter being about 5 mm. long. Since in this individual case there was no need for conservation of the bone, the laminæ and spinous processes of the sixth, seventh, and eighth dorsal vertebræ were removed. The dura was split, retracted by threads, the cord was slightly lateralized by passing threads about the posterior roots and gently using them as tractors, and the cord incision was made on each side with very little bleeding. The wound was closed without drainage and, in so far as it was concerned, the after-course was uneventful.

The approach to the cord is greatly simplified and expedited—although it was not done in this case—by the use of the Hudson trephine and the Cryer bone-cutting instrument. By the help of these tools it is possible rapidly and safely to obtain, by unilateral resection, preserving the spinous processes, an exposure adequate for many forms of dural and cord intervention. It would certainly be so for a limited section, such as was practised in this

case, or for section of posterior roots. Such exposure leaves little or no subsequent crippling so far as the spinal column is concerned and is much simpler than an osteoplastic flap. In case of unilateral pain, uncontrollable by other means, cord section on the side opposite to that in which the pain is felt and sufficiently high to allow for the decussation of sensory fibers should be adequate.

EXPERIMENTAL WORK ON THE FUNCTION OF THE ANTEROLATERAL COLUMN OF THE SPINAL CORD[1]

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The present investigation was undertaken at the suggestion of Dr. William G. Spiller, in order to determine what symptoms would be produced by destroying the anterolateral column, including Gowers' tract. In Gowers' original observation[2] he referred to a group of fibers situated in the anterolateral columns of the cord, which underwent degeneration from a lesion of the eleventh thoracic segment. He traced these degenerated fibers upward as far as the cervical region, and believed that they were concerned in the transmission of painful stimuli from the opposite side of the body.

Since that time innumerable clinical and pathologic studies seem to confirm Gowers' first impression. Experiments on animals, however, have not always lent support to this opinion, the results in some instances having been very confusing and even contradictory; indeed, Mott,[3] after cutting this tract in monkeys, concluded that its function was unknown. Bing,[4] experimenting on dogs, observed ataxia without disturbance of

[1] Read April 3, 1912.
[2] Diagnosis of the Diseases of the Cord, 1879.
[3] Brain, 1895, xviii, 1.
[4] Die Bedeutung der spinocerebellaren Systeme. Wiesbaden, 1907; Neurol Centralbl., 1912, p. 270.

cutaneous sensation, but in his cases, as in Marburg's,[1] the area destroyed was chiefly the dorsal cerebellar tract or close to it. Bruce and Schäfer[2] conducted similar experiments on monkeys in which only the ventrolateral region was destroyed; they not only found sensation entirely normal, but, unlike Bing, could not demonstrate any sign of ataxia, though paralysis did occur. On the other hand, Lewandowsky,[3] Rothmann,[4] and others rather positively state that section of Gowers' tract in dogs does produce a certain degree of analgesia.

In the following experiments our object was to completely destroy the anterolateral region, including Gowers' tract, and not to injure the area occupied by the dorsal cerebellar tract or any part of the gray matter. Dogs were chosen for purely extraneous reasons.

Under complete ether anesthesia an incision was made over two spinous processes and carried down directly to the bone; this incision was carried on as a subperiosteal resection of the processes and the laminæ of the vertebræ, until the cord was exposed for about 3 cm. The dura was slit longitudinally in the midline and retracted on either side; the cord was then rolled over as gently as possible until the line of insertion of the ligamentum denticulatum was identified, believing this to correspond quite accurately to the posterior limits of the anterolateral tract; a small knife was inserted immediately in front of that point and thrust obliquely forward, then cutting outward through the periphery of the cord. A similar incision was made at the corresponding point on the opposite side. There was little hemorrhage. The dura was closed with a fine silk suture and the wound repaired with catgut. Iodine and collodion were used for dressing. Wound healing was perfectly normal.

The dogs were carefully watched from day to day; generally the first observations recorded were made a day or two after

[1] Arch. f. Physiol , Suppl., 1904, p. 457.

[2] Quart. Jour. Exper. Physiol., 1910, No. 3.

[3] Handbuch der Neurologie, pp. 773 and 779.

[4] Berl. klin. Woch., 1906, No. 2, p. 47; ibid., 1901; Arch. f. Physiol., 1902, p. 153; Suppl., 1902, p. 440; Zeits. f. klin. Med., 1902, xliv, 183; Neurol. Centralbl., 1911, p. 1207.

operation, so that the effects of the operation itself might not be mistaken. Here we wish to point out one possible source of confusion. Anyone at all familiar with animals, particularly the laboratory dog, should not lose sight of the fact that their general conduct and intelligence influences the manner in which they react to stimuli of any kind; even in health this may differ very greatly. Each dog has his own peculiarity, and has a distinct individuality not unlike man. Many are extremely dull and apathetic and others high-strung, intelligent and active, and accustomed to responding quickly to the various stimuli originating through the association of friends and master. No doubt it was a thorough appreciation of these facts which led Lewandowsky and Kallisher to adopt their methods of training. Our dogs were not trained; nevertheless, a careful estimate of individual traits and temperament was constantly kept in mind. In considering disturbances of sensation these facts are of the utmost importance and have therefore been referred to in detail.

After a period of time the dogs were killed, and the brains and cords immediately removed and placed in Müller's fluid. The segment in which the incision had been made was mounted in celloidin and cut in serial sections in order to ascertain the exact limits of the area destroyed; sections were also made from each segment of the cord and parts of the medulla and pons, so that the course of degeneration could be followed.

In the following descriptions of cases the data have been given chronologically so as to show the course of the general symptomatology; then follows the microscopic study.

Dog 1.—Black and tan, short-haired male dog, weighing about 25 pounds. Operation, December 4, 1911. Cutting the anterolateral column of both sides in the seventh thoracic segment.

December 5: Incomplete weakness of both hind legs, more marked on right. Dog can stand, but often falls behind. After falling it can rise on hind legs without evidence of weakness, but is awkward and can only stand a second or two before again falling. It frequently assumes curious positions as if kneeling, or may stand with right foot flexed so that dorsum of foot touches the floor; sometimes sits with weight on one leg

which is crossed under rump. These positions do not seem to cause any discomfort, and appear to be from impairment of the sense of muscular position; it is very striking, and it even seems doubtful if there is any real motor weakness, the apparent weakness being due to uncertainty of ataxia. Sensation for pain tested with pin-point and with hemostats shows definite impairment; dog reacts quickly in front legs and shoulders, and very slowly or not at all on hind legs. Extreme heat gives the same results.

December 8. Sensory tests gave same results. Ataxia seems definite and very striking; no motor palsy.

December 10. Sensory tests the same. Dog can stand on all four limbs without evidence of ataxia or of motor weakness. Ataxia very striking when in motion, trunk and rump involved, feet frequently crossed and seem to be misplaced so that dog often falls; curious positions when at rest as before, very striking. Both sides alike, possibly right side more ataxic.

January 4, 1912, Bilateral ataxia of hind legs and rump very distinct in walking and running. Dog falls very frequently with hind leg. Curious position of legs seen less often than before. Painful faradic current causes slight or doubtful evidence of discomfort in hind legs and rump, but distinctly less marked than on front legs, where it is positively painful. When applied to soles of feet, hind legs are drawn away but not promptly, while on soles of front feet legs are drawn away quickly and with vigor. Application of hemostats caudal to site of operation is distinctly less painful than above operation scar, though when attention is directed to the procedure the dogs seem to feel some discomfort very occasionally. For extreme heat the same holds true.

January 11. Ataxia is gradually recovering; no other change.

January 18. The same as on January 11.

February 1. All sensory tests the same as before, but at times very confusing, depending very much on whether or not dog's attention is directed to what examiner is doing or to distant object. When attention is held by assistant then analgesia is very pronounced and unmistakable, but when attention is not diverted, sensation is affected, but not at all pronounced or even doubtful. Rapid examination might easily show that sensation was normal, but careful, frequent, and prolonged examinations make it unquestionably correct that there is incomplete analgesia, and thermo-anesthesia and pronounced bilateral ataxia of hind legs and no motor paralysis.

Dog. 2.—Black and tan, long-haired male dog, weighing about 25 pounds. Operation, December 5, 1911: cutting the anterolateral column of both sides at seventh thoracic segment.

December 6. Dog cannot stand or walk on account of inability to use hind legs. Tendon reflexes are equal and active. There is no spasticity nor do the hind legs feel exactly flaccid. Two sides are alike. Sphincters are not affected; no spontaneous pain. Application of hemostats over hind legs and also heated instrument seems to cause no discomfort; when it is applied to front legs dog reacts rather slowly, so that conclusions are unreliable.

December 10. Ability to use hind legs voluntarily rapidly returning. Dog makes effort to stand and walk; can rise on hind legs, but generally falls; muscular weakness very doubtful. Seems as if he had some loss of sense of muscular position; legs appear awkward. Pinching of skin over hind legs with hemostats does not seem to be appreciated; but when applied to front legs it is quickly recognized. Testing with heats perhaps the same but unreliable.

December 15. Loss of the sense of muscular position or ataxia very striking. No motor weakness. When standing at rest dog is inclined to lower head below line of shoulders, with hind legs somewhat farther apart than normal and slightly rotated outward; vertebral column shows slight tendency to arching in lower thoracic and lumbar region; this varies from time to time; the tail is held in what may be a purposeful position, straight out from the body, varying in its angle as position of legs is changed, but generally curved, tip turned downward or laterally and more or less rigid at base. The general position is very like that assumed by a dog standing on a swing in motion, attempting to retain balance. In walking or running he repeatedly falls with hind legs; feet are often crossed, but when moved are lifted clear of the floor, though not higher than normal. Rump sways a little. Dog often assumes unnatural positions, typical kneeling position, or stands on dorsum of foot, toes being in flexion, one or both hind legs may be crossed under rump when in sitting position. This does not seem to be in the least uncomfortable.

Pinching with hemostats and application of extreme heat over rump, hind legs and belly do not appear to be as distinctly or as quickly felt as when front legs are tested. At times there seems to be very decided analgesia, but a few minutes later dog seems to feel quite well; although when compared to front legs there is a decided difference in reaction time. The tendon reflexes are equal and active.

December 20. About the same as on December 15.

December 29. Ataxia is somewhat less marked. Dog does not fall so often in walking and running. Sensation the same as before.

January 4, 1912. Much the same as before. Application of painful faradic current gives exactly the same results as in first dog.

January 15. Sensory tests about the same. Analgesia seems definite at times, while at another examination it seems very doubtful. Ataxia

less marked; dog still falls when walking, but unnatural positions are assumed only very occasionally. In standing the position already described is still very striking.

January 24. Just the same.

February 6. Dog killed sixty-three days after the operation and the brain and cord removed for examination.

Conclusions. Ataxia very striking.

Impairment of sense of pain and for extreme heat and painful faradic current over lower half of body and lower extremities.

Microscopic examination showed that the anterior and lateral portions of the white columns of the cord had been destroyed on both sides. A line drawn through the posterior limits of this area would just bisect the central canal. Anteriorly it extended to a point which corresponded to the position of the most medially situated anterior root fibers. The ventral and dorsal cerebellar tracts were degenerated as far as the pons. In addition some of the degenerated fibers seen in the thoracic segments of the cord corresponded to the positions given by Edinger[1] for the spinotectal, spinothalamic, vestibulospinal, and spinovestibular fibers. In segments below the area destroyed there were a few scattered degenerated fibers situated in the extreme lateral periphery. The pyramidal tract and von Monakow's bundles did not seem to be injured. The posterior columns were intact.

In both dogs motor paralysis was noted immediately following the operations, but it disappeared rapidly and was replaced by very marked ataxia. Why motor weakness should occur at all is difficult to explain, for we believe that Gowers' tract is sensory in function and the microscopic examination showed that the pyramidal and von Monakow's bundles were not injured. It may have been from temporarily disturbed vascular supply.

Rothmann has proved that when both the pyramidal tracts and von Monakow's bundles are destroyed, either in dogs or in monkeys, the resulting paralysis may not be permanent. He was forced therefore to conclude that volitional impulses might be conducted from the cortex by some other route. In this connection Schäfer's experiments are important. He cut Gowers' tract in monkeys and obtained motor weakness, but without any evidence of ataxia or of cutaneous sensory disturbances. In order

[1] Bau der nervosen Zentralorgane. Ed. 8, 1911.

to explain the occurrence of paralysis he inclined to the opinion that voluntary impulses must be carried from the cortex down the cord by fibers which arise in Deiters' nucleus, and pass through the ventral and anterolateral columns near the periphery, and represent therefore a subsidiary motor pathway. It does not seem possible to make a complete section of the anterolateral tracts without destroying these long descending fibers; in our cases they must have been destroyed, yet motor paralysis was not permanent. Microscopic examinations of the segments below the level of section did reveal some evidence of scattered descending degeneration; the great majority of degenerations, however, occurred above the area destroyed and extended in the ascending direction.

Consequently Schäfer's[1] explanation does not seem to us entirely satisfactory. The experiments of Mott and Sherrington,[2] on the other hand, are most suggestive. They divided the posterior roots of the nerves supplying the brachial plexus in a monkey, and found that the arm was as much paralyzed for ordinary movements as in other cases in which the motor nerves themselves were cut. This they attributed to the cutting off of sensory impulses which pass from the muscles to the cortex. Bastian, referring to these experiments, suggested that in consequence of dividing the posterior roots there must have been a loss of tone in the neuromuscular apparatus. This seems to us the most probable explanation, for we know that the fibers of Gowers' tract represent the secondary continuations of some of the posterior root fibers, and it is probable that the function of one is represented in part by the other. Furthermore, it is known that the fibers of Gowers' tract end chiefly in the cerebellum.

Inasmuch as our dogs did not show the ordinary signs of spastic paralysis and the apparent motor weakness gradually subsided, and ataxia became more and more apparent, and finally entirely replaced disturbance of voluntary motion, we believe that motor paralysis in the true sense never existed. But on

[1] Jour. Physiol., 1899, xxiv, 23.

[2] Quoted in Schäfer's article.

account of a very profound derangement of the neuromuscular apparatus, together with impairment of cutaneous sensibility, a pseudoparalytic state or a true paralytic ataxia did occur, because sensory impressions which should pass through Gowers' tract had been cut off; and as the compensatory function of the remaining sensory tracts was being established the apparent paralysis gradually disappeared and ataxia which had previously been masked then became evident and remained permanent.

The character of the ataxia was similar to that described by Bing and Marburg, and, according to them, should be expected, as the spinocerebellar tracts are essentially the afferent connections between the cerebellum and spinal cord. It seems to us that the rubrospinal and vestibulospinal fibers may, as Bing has lately indicated, represent efferent connections between the cerebellum and cord and their destruction might be partially responsible for the extreme incoördination; however, proof is lacking.

In regard to the occurrence of cutaneous sensory disturbances there is considerable difference of opinion. Schäfer, Bruce, Bing and others were unable to demonstrate definite cutaneous disturbances. On the other hand, Rothmann, Schuster, Lewandowsky, and others all agree that Gowers' tract in the dog does convey painful sensations, though the posterior columns are also concerned. Why such contradictory views should be expressed we have no explanation to offer unless it is that the character of the dogs has not always been considered.

In concluding we feel certain that our dogs did have a definite though incomplete loss of cutaneous sensation for pain and for extreme heat, and very pronounced ataxia of the hind legs and rump. The fibers whose function it is to conduct painful sensations probably are connected with the thalamus and are situated close to the tractus spinocerebellaris ventralis, whose function is chiefly concerned in the regulation of purposive movements.

A YEAR'S WORK IN APPENDICITIS[1]

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It is an encouraging sign of the times when the number of operations for chronic appendicitis exceeds those for the acute disease. It is also far better for the patients, as I shall show.

This paper covers the work done at the German Hospital and the Children's Hospital of the Mary J. Drexel Home as well as that of my clinic at the University Hospital. During the past year 561 chronically diseased appendices have been removed without a death. In the same time there have been 356 cases of acute appendicitis with 8 deaths.

That the existence of chronic appendicitis is productive of symptoms no longer admits of question. The question of real importance is the diagnosis of the condition. Usually it is anything but difficult, necessitating only the patient's brief recountal of symptoms supplemented by palpation of the right iliac fossa; still, often enough, it may be a matter of the greatest difficulty. This is due to the facts that, first, chronic appendicitis does not always give symptoms referable to the right iliac fossa, and, secondly, that symptoms referable to the right iliac fossa are not always due to a diseased appendix. The train of symptoms set up by a diseased appendix may be referable to a distant point, most commonly the epigastrium. Stomach trouble often turns out to be appendix trouble. Pain in the epigastrium, distress after eating, nausea, belching, vomiting, the protean forms of indigestion may be caused by chronic appendicitis without symptoms

[1] Read May 1, 1912.

or palpable evidence of disease in the right iliac fossa. Certain of these cases, if watched closely, will have slight exacerbations of the local process, during which the diagnosis may become clear. In other cases the state of affairs will become evident only by abdominal section, when a negative examination of the upper abdominal viscera, coupled with the finding and removal of a chronically diseased appendix, followed by recovery from symptoms, will substantiate the diagnosis. Many other cases will present insufficient symptoms to warrant exploratory incision, and will drift along to spontaneous recovery or to confirmed dyspepsia, with involvement of the other organs of digestion.

I know of no way to diagnosticate chronic appendicitis without pain and local symptoms. Pain in some form or degree is our greatest reliance, yet it has become evident to me that chronic appendicitis is not always accompanied by pain. Since I have been removing many appendices as an incidental procedure when operating in the neighborhood it has been found by the pathologist that a large percentage of these appendices present unmistakable evidence of past or present inflammation. It would seem that local pain does not necessarily accompany inflammation, but is present only when colic is caused, when the intra-appendicular tension is raised by stricture or retention, or when the serous surface is involved in the inflammation. This is not surprising, in view of the observation of Lennander and others showing that the abdominal viscera themselves are quite insensitive to pain. In a few cases chronic appendicitis proceeds by slow stages to complete obliteration of the organ, which is Nature's method of spontaneous cure, without noteworthy symptoms of any kind and without any symptoms of the sort that we now recognize as pointing to the appendix. These remarks will illustrate the difficulty of recognizing existing chronic appendicitis, though it may be present in an advanced stage, and, if time permitted, I could cite many cases in point.

The second difficulty relates to symptoms simulating disease of the appendix. These may be due to movable right kidney, to renal or ureteral calculus, to cholecystitis, pancreatitis or

duodenal ulcer, to right-sided disease of the ovary or Fallopian tube, to bands or adhesions such as we are speaking of recently as Lane's kink of the ileum and membranous pericolitis, to an abnormally movable cecum, and, finally, to one of a number of very uncommon conditions, such as typhlitis, suppuration at the internal ring, etc.

I am led to speak of these conditions because of the fact that the operation of appendectomy for symptoms believed to be due to chronic appendicitis though almost uniformly successful in bringing relief does not in every instance do so. When symptoms persist after the operation they may be due to several causes: (1) Adhesions may be present either as a result of the appendicular disease, or the operation itself; (2) secondary changes in other organs may have occurred—for instance, there is reason to believe that cholecystitis and cholelithiasis and ulceration of the stomach or duodenum may depend in some obscure way upon chronic appendicitis; (3) the functional derangement, commented upon previously, when of long duration may have become engrafted as a habit upon the physiologic activities of the alimentary tract; (4) the diagnosis may have been mistaken.

The chief safeguard against mistaken diagnosis is a careful history and the following up by more extended examination of the leads that may be obtained.

Movable or diseased kidney, renal or ureteral calculus, upon careful inquiry will usually show in the history some abnormal character of the urine, of the act of micturition, or a characteristic type of pain. The floating kidney can be palpated and the presence of stone may usually be confirmed by the *x*-ray and cystoscopy.

It must not be overlooked, however, that chronic appendicitis may occasionally give pain referred to the genitalia or the leg. Cholelithiasis, cholecystitis, chronic pancreatitis, and duodenal ulcer, when they give rise to the typical symptoms, are in no danger of being mistaken for chronic appendicitis, but we have long since learned that they may give indifferent symptoms with no pathognomonic value, simple indeterminate dyspepsias, and when

this is true they are in danger of being mistaken for the less outspoken varieties of chronic disease of the appendix. Only those who never follow their diagnosis inside of the abdomen can harbor the delusion that these differentiations are always possible.

Disease of the right Fallopian tube or ovary will also, as a rule, give symptoms that are definitely related to the sexual history and function, and the condition may be demonstrated, though not invariably so, by examination. Usually the signs and symptoms are bilateral, tenderness is lower in the pelvis, nausea, vomiting and digestive derangements are less frequent, and the regularity of exacerbations at the menstrual period is very significant. Here we must mention that chronic appendicitis may give pelvic symptoms in the absence of pelvic disease, and that the two conditions may coexist. With a history of pelvic disease and a negative examination of the internal genitalia the possibility of chronic appendicitis should always be strongly considered.

The attention which has recently been focussed upon the presence of abdominal bands and membranes in the region of the cecum has resulted in demonstrating that these formations are not infrequent, and that they may upon occasion give rise to symptoms which are very like those of chronic appendicitis. The same is true of an abnormally movable cecum, which has been emphasized by the Germans, and recently by Sailer in this city. Commonly the obstructive element and intestinal stasis, with its train of symptoms, are more likely to be prominent in these conditions. There is no way of differentiating with certainty before operation and inspection. Much is claimed for the *x*-ray in the diagnosis of these conditions, and doubtless there is a percentage of cases in which help can be obtained. Skiagrams of abdominal viscera are, however, apt to be misleading, and particularly so the further away they are from the orifice through which is introduced the bismuth or other mixture that is needed to outline the viscus. Too much importance should not, therefore, be attached to skiagraphic diagnosis.

It remains for the surgeon to satisfy himself at the time of operation that the appendix is such as to account for the

symptoms. This he should do by inspection or palpation before concluding the operation.

I am not greatly impressed with the cry that has at times been raised as to the unsatisfactory results of appendectomy in many cases of supposed chronic appendicitis. To me it has seemed that no operation is more uniformly satisfactory to the patient from the standpoint of relief of symptoms. The elimination of unsatisfactory results depends more upon the care with which the diagnosis is made than upon any other factor, and since we have recognized that many atypical symptoms may be due to chronic appendicitis and are operating for their relief, it is well to utter a word of caution that in the cases which give no history of exacerbations that can be easily recognized as appendicitis special care must be used in assuring ourselves that we are dealing with appendicitis and not with one of the conditions that may simulate it.

In acute appendicitis the situation is the reverse of that in chronic appendicitis in respect to diagnosis and treatment. The acute forms of the malady are, as a rule, easily recognized by one of experience in abdominal disorders, while the treatment is difficult, and methods vary widely in skilled hands, and not a few important questions yet await settlement.

Perhaps the most important question that concerns the internist and practitioner, as well as the surgeon, is the use of purgatives during the acute stage of the disease. It is not long since we all thought that a purge should be administered in the initial stages of the disease, but we have come to realize that the contents of the bowel play practically no part in the continuation of the disease, while the peristalsis induced by purgation has proved to be a most unfavorable factor, defeating the end and principle upon which our treatment is based—namely, anatomic and physiologic rest. The purgative is more deadly than the scalpel. Of this I have long been convinced, but, with a view to obtaining exact figures to support my contention, I have had the year's work analyzed with respect to this point. Among 259 adults there were 79 that had a history of being purged before admission. Of these 79 cases all save 2, at the time of opera-

tion, showed appendices that were either perforated, gangrenous, acutely ulcerated, or surrounded by an abscess. The remaining 2 only were simple catarrhal inflammations. Of the 7 deaths occurring in the series 5 were purged before admission. Of those drastically purged 97.5 per cent. showed the severest kinds of appendicular disease. The exact figures are here given:

Pathologic lesion of appendix: Perforation, 30; gangrene, 9; acute ulcerative and suppurative, 23; peri-appendicular abscess, 15; acute catarrhal, 2.

Among 100 children the same rule holds. There were 56 that had been vigorously purged. Of these 86 per cent. showed the more serious lesions, such as gangrene, perforation, abscess, etc. Twelve cases had diffuse peritonitis, all of which had been purged before admission. The table is as follows:

Pathologic lesion: Perforation, 22; gangrene, 15; acute ulcerative, 6; peri-appendicular abscess, 4; acute catarrhal, 8; indefinite, 1.

In this series there were 44 acute cases that were not purged, and in this group there was but 1 case of perforation, 8 cases of gangrene, and no peri-appendicular abscesses. The remainder showed acute catarrhal or ulcerative inflammations which were strictly confined to the appendix. Consider these figures and tell me whether the mortality of appendicitis is medical or surgical. The time is not far distant when the man who uses a purge in acute appendicitis will lose his standing in the profession as well as his patient. In these cases the enema should always be used when it is desired to move the bowels, but in general less attention should be paid to evacuation of the bowels and more attention to placing them at rest and to the abatement of peristalsis.

Special care is necessary in the treatment of appendicitis in children. It is by no means uncommon, but is frequently overlooked on account of the prevalence of colics and gastro-intestinal derangements among the young. Unquestionably fermentation and gastro-enteritis often are best treated by purgation, but before beginning this treatment appendicitis should be carefully excluded. In general, the disease in children is identical in its

pathology and clinical manifestations with that seen in adults. We do find, however, that the pathology not infrequently moves with startling rapidity; gangrene, perforation, and diffuse peritonitis, all within the course of twenty-four hours or less, not being exceptional. On the other hand, children resist continued sepsis more vigorously than adults. From the operative standpoint we find that children are more subject to shock, but unless this ends fatally their recuperative power rapidly asserts itself.

The medical treatment of acute appendicitis, in my opinion, consists of the following:

1. Diagnosis.
2. Avoidance of purgative or laxative medicine.
3. The institution of anatomic and physiologic rest.
4. Immediate preparation for removal of the appendix.

Anatomic and physiologic rest are secured by the following measures:

1. Complete rest, preferably in the sitting posture, to aid in the gravitation of exudates, if any, toward the pelvis.

2. Absolute prohibition of anything by the mouth, including water and ice, as well as nourishment, in order to quiet peristalsis.

3. Gastric lavage to relieve the stomach of accumulated fluids, preventing, in large degree, toxemia, vomiting, and peristalsis.

4. The constant instillation of normal salt solution by way of the rectum according to the method devised by Murphy. This diminishes thirst and supplies the need of water.

5. Ice-bags, over the abdomen to quiet pain, to assist in keeping the patient quiet, and, more particularly, to discourage meddling examinations.

I have said nothing about drugs, for they are not necessary. Usually they are a disadvantage. Gastric sedatives are worse than useless. Morphine, if given indiscriminately, masks the symptoms, interferes with the phagocytic activity of the leukocytes, and inspires both patient and physician with false confidence. The pain of appendicitis is not often so great as to require morphine, but if necessary it should only be given in a minimum dose after decision for operation has been made. Under the above treatment

the pain abates and no anodyne is needed. In the German Hospital and the Children's Hospital of the Mary J. Drexel Home it is never found necessary to use opiates prior to operation, and I cannot believe that the disease is so different elsewhere. It is far better never to use them than to use them once too often. In the rare cases where operation is impossible they may, of course, be given in accordance with the indications of extreme restlessness and pain.

This series bears out what we already had established regarding the liability of recurrence of attacks, for of the 359 cases 170, or practically one-half, had had previous attacks. This means that 170 individuals, either of their own volition or more commonly upon the advice of their physician, in order to avoid operation had bartered the operative mortality of uncomplicated cases, in this series nothing and never great, only to be obliged to accept later the risk of the general mortality of this series—4.2 per cent. Three of these patients, all of whom had been drastically purged, died unoperated, being admitted in a state of advanced diffuse peritonitis that precluded either operation or improvement. This leaves an operative mortality in all acute cases of 2.7 per cent. in adults and 1 per cent. in children. Nor is this the only difference, since recovery in uncomplicated cases was almost without exception speedy and without complication, while many who recovered after acute attacks did so only after a battle with sepsis, which depleted their physical powers, wasted their resources, and, at times, left impairments, such as fecal fistula, phlebitis, adhesions, and ventral hernia, while one case developed postoperative insanity.

In females the obligation to advise early operation is pressing because of the danger of tubal involvement. In 7 cases I found the appendix tied up in an inflammatory mass with the right tube and ovary, and in 17, tuboövarian disease coexisted.

As a cause of sterility appendicitis possesses considerable importance and is not an infrequent cause of pelvic inflammation in young females. From the standpoint of mortality, however, the female sex possesses an advantage, since they resist peritoneal,

and especially pelvic, inflammation better than the male. Of the 7 postoperative deaths among adults all were males.

A factor over which we have no control, but which greatly influences the prognosis, is the situation of the perforations. Those which occur at the base rapidly give rise to a peculiarly virulent type of peritonitis and are likely to cause death. The simple reason is to be found in the fact that a hole in this location allows the teeming bacterial life of the cecum to pour out into the abdominal cavity in overwhelming dosage. Another highly dangerous form of the disease is that in which the appendix, lying behind the cecum and ascending colon, causes retrocecal suppuration, and infection of the cellular retroperitoneal tissues, where drainage is difficult and absorption rapid. Infections in the central region or enteronic area among the coils of small intestine are also difficult to handle and exceedingly likely to cause diffuse peritonitis. The most favorable areas from the standpoint of treatment are the marginal or lateral and the pelvic zones. These locations are influenced chiefly by the location of the appendix, which we are unable to influence or determine beforehand. Consequently our only resource is prevention by removal of the appendix at the outset before it can induce suppuration of any area.

In regard to the treatment of peri-appendicular abscess I am still a believer in the method of extraperitoneal approach when it is feasible. Many abscesses have, as a part of their retaining wall, the parietal peritoneum of the right iliac fossa or the loin. When Nature has thus excluded the infectious area from the general cavity I would not think of attacking the collection transperitoneally by tearing down the defensive adhesions between the intestines and omentum. Much simpler and much safer, in my opinion, based on results, it is to open directly into the abscess through the abdominal wall. There are many cases in which the abscess is not in so favorable a situation and must be attacked transperitoneally. In this case protection to the general cavity must be secured as well as may be by the disposal of gauze packs during operation and the arrangement of drainage when the infected area has been dealt with. Even here I do not break up

adhesions unnecessarily, but only enough to deal thoroughly with the abscess cavity. The proposal of some surgeons to relieve all the adhesions is contrary to reason and results. It is not the breaking up of adhesions, but the institution of drainage that cures. It is a general rule that we cure best when we work with Nature rather than by flouting her efforts and substituting our own works for those of our Creator.

As a corollary to this attitude regarding peri-appendicular abscess I take issue with those surgeons who never leave the appendix.

In the 356 operated cases of acute appendicitis upon which this paper is based the appendix was not removed in 15 cases (4.2 per cent.), and I have not yet had cause to regret leaving it in any of these cases. True, there is a slight danger of recurrent attacks in such a case, but this can be obviated by a second operation for removal of the appendix at the same time that the operation for the inevitable ventral hernia is performed. The appendix need not be left in any case that can be so securely closed as to obviate incisional hernia, so that a second operation is not often avoided by those who insist upon its removal. To remove it certainly does not assist in recovery from the attack itself, while the breaking up of adhesions and burrowing about in the inflammatory exudate in the effort to locate a buried appendix is certainly dangerous. Even were it possible, by removal of the appendix, to secure better closure of the abdominal wall, I would still not do it in certain cases, for it has always been my belief, frequently stated, that a living patient with an appendix is better than a dead patient without an appendix. I always make an effort to find the appendix, and usually am successful without the infliction of much damage; but there is a limit beyond which I will not go even in the interest of thoroughness, much preferring the interest of the patient.

Another subject for serious disagreement among surgeons relates to the time of operation in cases of spreading peritonitis. There can be no disagreement as to the advisability of operating early. When the disease is still confined to the appendix,

even in acute appendicitis, the technique is as simple and the results just as satisfactory as in chronic disease. The difficulty and the danger arise when the inflammation has passed beyond the confines of the appendix, whether by perforation or by extension through its walls without gross perforation. Whatever the cause of the initial lesion of the appendix, the peritonitis resulting from it does not present an extraordinarily diverse flora. Thus in the series under consideration in the cases of peritonitis we found the following organisms: Bacillus coli, 171; bacillus pyocyaneus, 7; bacillus fæcalis alcaligenes, 5; staphylococcus albus, 5; streptococcus, 2.

The preponderance of colon bacillus is natural in infections derived from this region of the alimentary tract, and gives considerable uniformity to the picture of appendicular peritonitis. Bacillus pyocyaneus is somewhat more dangerous than the colon bacillus. Bacillus fæcalis alcaligenes runs practically the same course as the other members of the colon group.

The Staphylococcus albus, which has been so loudly proclaimed by Dudgeon and Sargent as the friend of the peritoneum, "the first to come and the last to go," we do not find so uniformly, though we have made many cultures in exactly the manner prescribed by these authors. We can confirm their results, however, in respect to its relative harmlessness.

The streptococcus of appendicular inflammation does not appear to possess the same pathogenicity as is usually associated with the streptococcus elsewhere. This may be due to the fact that the streptococcus fecalis is different biologically from the streptococci found in the sputum, in erysipelas, or in uterine and cellular infections. It reacts differently toward the test sugars, and may well do so in the human body. Or the difference may be due to the location, the better opportunity for drainage, or to some other clinical reason. At any rate, streptococcic peritonitis of appendicular origin is not so fatal as streptococcic peritonitis secondary to other intra-abdominal lesions, though it is more serious than colon infections.

Now, when these organisms reach the peritoneal cavity the

result depends upon three factors: (1) the virulence of the organism, (2) the resistance of the body, local and general, (3) the dosage or amount of infection. Over the first two factors we have little control. Our influence is to be directed chiefly upon the last factor. By early operation we can rescue the peritoneum before the infection becomes too great to counteract. Just as our laboratory animal will react from 0.5 c.c. of a virulent culture of colon bacilli when 5 c.c. would cause a fatal peritonitis, so will our patient recover from early peritonitis and a limited infection when an additional amount will give rise to a diffuse peritonitis. In this early stage of diffusing peritonitis, which is still local though unconfined, we should operate at once. Clinically, this stage extends well up toward forty hours after the onset of the disease, though there are cases in which the process is more rapid than this. If we remove the appendix, prevent it from pouring out more infective material into the peritoneal cavity, the dosage of bacteria is not yet sufficiently great to set up a diffusing peritonitis, but is speedily overcome by the resisting mechanism of the body.

If we fail to operate in this early stage, peritonitis results, and, if so rapid and virulent that it marches ahead of the formation of plastic peritoneal adhesions, which seek to confine it to its origin, we have a diffuse peritonitis. This peritonitis, moreover, may be no more dependent upon the presence of the infecting focus —in this case the appendix—than are the flames which devour a house dependent upon the match which set it afire. The disease is peritonitis and not appendicitis. The fireman must direct his hose against the house and not turn his chemical extinguisher against the match. Can we treat diffuse peritonitis by operation? From an experience covering the whole development of the modern treatment of peritonitis, I say, No. I have washed, wiped, drained with gauze and with tubes, employed antiseptics, and run the gamut of active procedures proposed for treatment. I can only echo the words of an English surgeon that "the more thorough the operation the more quickly the patient dies." After reducing the treatment to simpler and simpler lines, with corresponding

improvement in mortality as each successive procedure was abandoned, I have come to the conclusion that no operation should be done in the presence of diffuse peritonitis, and have in this way achieved better results than by any form of operation, however complicated, however simple. Remember, that these remarks do not apply to local peritonitis, whether confined or unconfined. They apply only to those cases, usually of more than forty hours' duration, in which the clinical evidence of widespread peritonitis is present. Such cases appear generally sick, the face flushed, respiration chiefly costal in type, the temperature elevated to 102° F., though it may be normal. The abdomen may be beginning to distend, the tenderness is general, though usually most marked in the region of the appendix. Peristalsis is brief and tinkling constipation absolute or, at least, obstinate. Such a patient is not a subject for operation. We cannot drain the entire abdominal cavity, and to remove the appendix, though that were desirable could we "wish" it out, does not aid the body to overcome the infection in the outlying areas where the conflict is being waged that determines the life or death of the patient.

Operation, with its depression of the vital powers and resistance, will tip the scale against the patient. Masterly inactivity, with appropriate treatment, will tide them over the crisis, allow victory in the remote areas of the peritoneum, and localize the infection in the neighborhood of the appendix, where it may be dealt with to greater advantage and with every prospect of success. The treatment is the same as that already spoken of as anatomic and physiologic rest. If we treat our patients in accordance with these principles, it is true we shall still have a mortality in dealing with this grave disease, but it will be chiefly the mortality of weakness, of cardiac, pulmonary, or renal complications, and not that of peritonitis, our greatest foe. In this series I lost 3 cases from peritonitis, which I fear were due to inability to apply correctly these principles. The remainder died of cardiac and pulmonary complications.

In conclusion I would cite the collected data upon the entire series.

ACUTE APPENDICITIS RECORDS OF THE GERMAN HOSPITAL, 1911

Number of cases operated		256
Recovered		249
Died:		
Pneumonia	3	
Peritonitis	3	
Acute dilatation of heart	1	
Number of cases of death without operation		3
Number refusing operation		15
Percentage of deaths		2.7
Females		102, or 40%
Males		154, or 60%
Of the 7 postoperative deaths all were males.		
Number of cases of peritonitis		140, or 54.68%
Number of cases having repeated attacks		108
Number having localized peritonitis (on admission)		60
Number with diffuse peritonitis (on admission)		41
Appendices removed		245, or 95%
Appendix not removed		11, or 4.3%
Number of cases drained		118, or 45.6%
Number of cases giving a history of being purged before admission		79
Pathologic lesion:		
Perforated or self-amputated, etc.		30
Gangrenous		9
Abscess		15
Acute ulcerative, exudate, etc.		23
Acute catarrhal		2
Cultures were taken in 179 cases and the following organisms isolated:		
Bacillus coli		120
No growth		44
Bacillus pyocyaneus		6
Bacillus fæcalis alcaligenes		4
Staphylococcus albus		3
Streptococcus		2
Complication:		
Broken-down wounds		37
Superifical		17
Deep		20
(Of these 4 were with salpingitis, and 1 with tuberculous lymphangitis).		
Fecal fistula		14
(Of which 9 were purged before admission).		
Ulcerated cecum		4
Bronchitis		4
Pneumonia		4
Subdiaphragmatic abscess		6
Secondary collection		3
Tuboövarian disease		17
(Of these 7 were with the appendix tied in the mass of the right ovary, etc.).		
Congestion of the lung, bronchopneumonia (?)		2
Intestinal obstruction		2
Pleural effusion		1
Acute cardiac dilatation		1
Phlebitis		1
Urinary fistula		1
Epididymitis		1
Postoperative insanity		1
Lane's kink		1

Acute Appendicitis Records of the Children's Hospital, Mary J. Drexel Home, 1911

Number of cases operated	100
Recovered	99
Died	1
Number of cases refusing operation	3
Percentage of deaths	1%
Females	39, or 39%
Males	60, or 60%
Number of cases having previous attacks	62
Number of cases distinctly localized to appendix (not peritonitis)	12
Localizing number of cases having cloudy fluid	17
Localizing number of cases having diffuse peritonitis	32
Number of cases having localized abscess (mass)	24
Number of cases subsiding (not peritonitis) (on admission)	8
Number of cases subsided (not peritonitis) (on admission)	7
Number of cases with appendix removed	96, or 96%
Number of cases appendix not removed	4, or 4%
Number of cases drained	53, or 53%

Condition of Appendix in Cases Purged

Number of cases giving history of being purged before admission . . . 56

Eighty-six per cent. of the 56 cases purged were of the gangrenous, etc., nature.

All but 8 cases being either gangrenous, perforated, etc., these being classed as acute.

Acute inflammation confined to appendix	8
Gangrenous	15
Perforated	22
Abscess	4
Acute, ulcerated, exudate, etc.	6
Indefinite	1

Twelve of these cases had diffuse peritonitis—classified under gangrene or perforated appendicitis, all of whom were purged.

Condition of Appendix in Cases not Purged

Of these there were 43, 8 of which were gangrenous; 1 of which was perforated, or 14 per cent.

The rest showed ulceration of mucosa or acute catarrhal inflammation.

Cultures Were Taken in 76 Cases and the Following Organisms Isolated

B. coli	51
Staph. albus	2
No growth	21
B. pyocyaneus	1
B. fecalis alk.	1

Complications

Abscess between layers of mesentery	2
Mesenteric lymph glands enlarged	1
Secondary collection, glands of mesentery	1
Fecal fistula (all were purged)	5
Ulceration cecum	1
Influenza	1
Nephritis	1
Lane's kink	1
Postoperative pneumonia	3
Multiple localized abscesses at operation	3
Pelvic phlebitis	1
Suppurating mesenteric glands	1
Toxic diarrhea	1
Broken-down wound	1
Twisted ovarian cyst, gangrenous, with appendix adherent to it	1
Bronchitis	1
Subdiaphragmatic abscess	1

DISCUSSION

Dr. John B. Roberts: Dr. Deaver has this evening taken a position almost exactly in accordance with my own far less experience. I agree with him that it is not always right to search for and take out the appendix in cases of abscess. The difficulty of diagnosis has at times been great for me; and I feel gratified that a man of so much more experience agrees that it is sometimes, particularly in chronic cases, exceedingly difficult to tell whether the appendix or some other abdominal structure is the offending organ. I have thought that I have sometimes found in acute appendicitis a change in the percussion note or auscultatory sounds at the base of the right lung. I always auscultate the chest in appendicitis and never trust to my assistants. There may be loss of resonance, the presence of rales, or some change in the respiratory murmur. Perhaps these signs are due to the fact that the diaphragm on the right side becomes rigid, because its full movement would increase cause of pain. It may be that thus a tension like that of the abdomen in the iliac region is developed. As a result slight congestion of the pulmonary tissues may occur. Such a thoracic sign may only mean that there is an acutely painful inflammation in the infradiaphragmatic organs, which prevents full inflation of the right lower lobe of the lung. In a recent case of appendicitis I was interested to find what I at first throught was a small amount of congestion in the right lung. Operation revealed an old appendicitis with adhesions. I removed the appendix. There were no symptoms after the operation from the wound; but there very soon developed acute pain in the chest with congestion and bronchitis. I

have been familiar, as have other medical men, with the fact that pneumonia and other pulmonary processes do give pain in the abdominal region similar to appendicitis. This case seemed to be possibly an example of a double infection; namely, in the imprisoned appendix and in the right lung.

Dr. Richard H. Harte: It is interesting to note how teachers have changed their minds upon this interesting class of surgery. It is not long since the ordinary medical man was censured severely if he had not thoroughly purged his appendicular cases with castor oil or salines. Dr. Deaver, if I remember, was at one time a strong advocate of this line of treatment.

The statistics are most interesting, and as compared with those of fifteen years ago are vastly better. The casual observer would infer that this is largely due to the increased skill of the surgeon of today, but it must be borne in mind that the medical side of the profession is a great factor in helping our results, as cases are recognized much earlier than formerly and are immediately hurried to the hospital, and thus come into the hands of the surgeon in much better condition.

As we all know, if we can deal with these cases immediately the surgical risk is enormously reduced. There is no man in Philadelphia to whom we are more indebted than to Dr. Deaver for persistently keeping this matter before our minds. He has been distinctly a leader in this field, yet when we compare the statistics of the German Hospital with those of other institutions we cannot but admit that they are very gratifying.

Dr. William L. Rodman: I regret that I came in late and did not hear the first part of Dr. Deaver's paper. I have heard enough, however, to convince me that his views are entirely in accord with my own. I think that practically every surgeon will agree with Dr. Deaver regarding the harmfulness of purgation in appendicitis; also that in abscess cases entrance should be made directly into the abscess rather than transperitoneally. Such route is justified only in very exceptional cases. In the third place, I have always felt that it is very dangerous to search for the appendix in the majority of cases of abscess. In these cases it should not be removed unless it can be found after a very limited or reasonable search. I am very sure that if I had suppurating appendicitis and was about to be operated upon I should say that my appendix should be let alone and that I would take care of it at a subsequent time.

I am thoroughly in accord with Dr. Deaver's position as to the propriety of operating in these cases of rapidly spreading peritonitis. I believe it is the greatest possible mistake to operate in these cases. From the day when Dr. Ochsner read his epoch-making paper at St. Paul in 1901, I have followed his line of treatment, keeping, as Dr. Deaver says, even a drop of water from the stomach, and waited until the peritoneal

symptoms have sharply subsided before operating. Like Dr. Deaver, I have my doubts about general peritonitis being cured in the large number of cases reported. It is easy to call a case one of general peritonitis, but that does not make it so. It is questionable whether *general* peritonitis is ever followed by recovery. I think a great deal of harm has been done by some very high in authority in leading the general profession to think that these cases of general peritonitis can be saved by the surgeon at the last moment. Such statements cause delay in these cases and prevent early operations which are practically always safe. Dr. Deaver has proved his conclusions by incontestable statistics.

Dr. M. Howard Fussell: I might say a few words from the standpoint of the internist in regard to appendicitis. It takes more courage perhaps than one can estimate to say anything that appears to be at all in contradiction to what Dr. Deaver says. We all know that Dr. Deaver has taught us more about appendicitis, has taught me, at any rate, than any other one man in Philadelphia. He taught me to operate early, at the very earliest moment, and I am afraid that the position he takes tonight—while to himself and Philadelphia surgeons, is doubtless perfectly justifiable, and while probably his results are better than when he operated upon every single case he saw—that these conclusions will be misread by the general practitioner of medicine. However justifiable Ochsner's views are in regard to waiting in certain cases of appendicitis, they make the ordinary practitioner wait too long, and he will not send his cases in the first twenty-four or forty-eight or seventy-two hours. I am speaking from what I know. I have had quoted to me that Ochsner says you must not operate early. I do wish that Dr. Deaver would say definitely to the general practitioner that which he has already said that the case must go early to the surgeon for operation in all cases, except perhaps where there is widespread peritonitis. Certainly since I have been following that rule as I was taught by Dr. Deaver, my mortality has been practically *nil*. When I waited to see whether or not the patients would have abscess many of them died. The thing that must be impressed upon the general practitioner is that he is to operate when he sees his case. He is not to wait for anything to happen, but he is to get the surgeon there and allow him to use his good judgment. I saw a case not long since that had been treated for dysentery for eight days. Even that case was not operated upon by the surgeon at the time that he saw it, but was treated by Dr. Ochsner's method and operated upon some ten days or two weeks, and at the time or after operation a local abscess was found. It seemed to me that the time to have operated on that case was when the surgeon saw it. I don't believe any living man can tell what condition the appendix is in underneath the belly wall. A patient may have a few symptoms and have a gangren-

ous appendix. Night before last I saw a woman at midnight with pain in the left side and no resistance on the right. Early next morning she was operated upon and a gangrenous appendix was found. No one could have told the condition of the appendix before operation. Therefore, I believe we ought to impress upon the general practitioner that the thing to do is to send the case to the surgeon immediately, and that in the great majority of cases they ought to be operated upon at once.

Dr. A. P. C. Ashhurst: No doubt Dr. Deaver and Dr. Fussell are right. The mortality does not come from appendicitis, but from complications. If the appendix is removed before complications occur the patients get well. If operation is delayed until there is abscess or the appendix becomes gangrenous many patients will die. If the physician would send patients to the hospital as soon as they are sick and the surgeon would go and see them and take out the appendix at once, whether it was at 2 A.M or 12 noon-day, many cases would be saved. Immediate operation in early cases saves the patients the discomfort and tedious convalescence often accompanying a drained wound.

Regarding the question of abscess, I think there are still a few surgeons, Dr. Murphy among the number, who do not like to cut directly into the abscess through the abdominal wall, unless the abscess is very large and almost ready to open. If you open through the peritoneal cavity, and isolate the abscess with gauze packs, you can evacuate it at leisure, find and remove the appendix, and close the greater part of the wound, and there will be slight risk of postoperative hernia. If, on the other hand, you go into the abscess directly through the abdominal wall, although you make a large incision, it is funnel-shaped, and you have to work in a confined space at the bottom of a deep wound. Often you do not find the appendix and a second operation is necessary. In any event the large wound must be left open almost in its whole extent, and postoperative hernia is the rule.

Dr. Deaver (closing): I am glad that practically all surgeons are agreed upon this question. In regard to the treatment of abscess, different surgeons take different views. There are different methods of getting patients well. Personally, I believe with Dr. Rodman that when we are fortunate enough to be able to deal with abscesses extraperitoneally we should do so; however, this is not possible in the majority of appendicular abscesses. Dr. Murphy, as Dr. Ashhurst says, takes exception to this method. Doubtless Dr. Murphy's results are as good as mine, perhaps better. I quite agree with Dr. Fussell, and I can fully understand how general practitioners fail to interpret properly the so-called Ochsner treatment or the let-alone treatment. If they have put a wrong interpretation upon it, a great mistake is made in not operating early. Widespread peritonitis is a different proposition.

DEATH IN TWO CASES OF OPERATIVE FIXATION OF FRACTURE OF THE FEMUR[1]

By JOHN B. ROBERTS, M.D.

The conversion of a closed fracture into an open one, for purposes of treatment, should be restricted to those lesions in which subcutaneous reduction of the fragments is otherwise very difficult or the maintenance of coaptation, previously obtained under anesthesia, unsatisfactory. An unknown proportion of fractures should be subjected thus to incision, in order that proper replacement of the fragments may be obtained and direct fixation accomplished. This method has been advocated by me in various papers on fractures published since 1885.[2] It, therefore, will be understood that I am not opposed to the operative treatment of closed fractures in selected cases. There is, however, at the present time such an unwise advocacy of the open treatment of fractures that records of fatalities occurring from this method of treatment should be published. I therefore report two deaths following operative fixation of fracture of the femur, with the hope that other surgeons may be influenced to do likewise.

The extensive urging of the operative management of these injuries, as an almost routine method, will lead to many fatalities in the hands of inexperienced operators. Many fractures of the bones of the upper and lower extremities are curable by non-opera-

[1] Read May 1, 1912.

[2] Trans. Amer. Surg. Assoc., 1885, pp. 6 and 105; ibid., 1892, p. 58; Annals of Surgery, April, 1895, p. 457; paper read at the British Med. Assoc., 1898, published in Phila. Med. Jour., September 24, 1898, p. 621; Modern Treatment of Fractures, D. Appleton & Co., New York, 1899; Manual of Modern Surgery, Lea Brothers & Co., Philadelphia, 1899, pp. 364 and 369.

tive means, provided that the surgeon has anatomical and mechanical knowledge. Such knowledge, combined with diligent attention to the treatment subsequent to the reduction of the fracture, will secure a satisfactory cosmetic and functional result in most closed fractures.

The great activity in the operative treatment of closed fractures, which has developed within recent years, is due in part to the study of the broken bones by the means of the *x*-ray. This method of examination has vastly increased our pathological knowledge of bone injuries. It has, however, created a tendency to make the attending physician or surgeon neglect other clinical methods, which were formerly used to obtain a proper knowledge of the condition of the injured bones.

Other factors encouraging more frequent operative attacks have been a general recognition of the safety of aseptic wounds, and the desire to avoid frequent visits and to lessen the time during which the activity of the patient should be restrained. Surgeons have become annoyed at the necessity for frequent adjustment of splints and apparatus, which is demanded in the treatment of fractures of the extremities not subjected to operative management. An undue reliance on the supposed truthfulness of even carelessly taken *x*-ray plates and an indolent neglect of fracture, treated by external appliances, have given rise to a belief that many fractures are curable only by exposure of the bones, and fixing the fragments with screws, wires, or metal plates. These operations are now being attempted by men who have not had sufficient surgical experience to be entitled to confidence when they attack deep-seated injuries of the skeleton. Good results with less risk to life may often be obtained by less dangerous methods.

The determination of whether a fracture should be subjected to operation by studying *x*-ray plates alone, leads at times to unnecessary operations. *X*-ray plates of fractures are of real value only when carefully and properly made by an expert radiologist, who is also somewhat acquainted with the surgical problems demanding solution. The plates may easily deceive the surgeon by giving an erroneous impression of the osseous relations. They

should be used only as collateral evidence in connection with other diagnostic and therapeutic methods at the disposal of the surgeon. Stereoscopic plates, or else plates taken in two planes at right angles to each other, may be required to furnish the information which the surgeon needs. They should be made before the fracture apparatus has been applied and also while it is being worn, if they are to be used to disprove the probable success of the non-operative treatment.

It seems to be often forgotten by surgeons that no accurate determination of the degree of shortening in fractures of the extremities is possible, unless there exist previous accurate measurements of the two extremities before the fracture occurred. Asymmetry in the length of long bones is a very common condition. I proved this in 1878[1] by measuring the bare bones of skeletons having normal femurs. This inequality in length has been repeatedly shown since that time by other investigators. To ascribe to imperfect treatment, non-operative or operative, a moderate degree of shortening in a leg or arm after fracture is a mistake, unless the exact equality in length previous to injury has been established. This, naturally, can scarcely ever be done in practical surgical work.

One sees reports of fracture of the femur treated by operation, because of the asserted inefficiency of its prior non-operative treatment. In some of these cases the prior treatment apparently has been conducted without giving the patient the benefit of strong continuous traction and countertraction by Buck's method. The muscles of the thigh are so powerful and usually cause such overlapping of fragments, when the femoral shaft is broken, that I doubt whether a surgeon is justified in adopting the open method unless he has first employed, for a couple of days at least, continuous traction and countertraction with from 15 to 40 pounds by the Buck method.

When a fracture of a long bone cannot be satisfactorily reduced, or when, if it can be reduced, maintenance of the coaptation is

[1] Philadelphia Medical Times, August 3, 1878.

impracticable, it is proper for a trained aseptic surgeon to operate. It is essential, however, that he or a similar expert have charge of the after-treatment. The operation should be done only when asepsis can be obtained, and be maintained after operation; and it is important that the surgeon and the attendants have expert knowledge and be willing to give the time to avert any secondary septic symptoms.

Fractures of the shaft of the femur just below the small trochanter, very oblique fractures of the shaft, and fractures at the lower end of this bone demand operative fixation with more frequency probably than other fractures of this or other long bones.

The non-operative method is usually sufficient to gain a good result in other fractures of the femur and in fracture of other bones of the extremities, provided that the surgical attendant knows the anatomy of, and the mechanical problems presented by, the injury, gives proper attention to the case, and supplements the want of knowledge of the other attendants upon the patient by his own surgical acumen and vigilance.

The operative treatment of fracture of the femur is more difficult than the operative treatment of fractures of the bones of the leg or of the upper extremity; but in selected cases it may be required. In some other cases it may be preferable to non-operative treatment, if the patient can be given the benefit of the proper environment and skill.

Direct fixation by operation of fractures of the large and deeply situated bone of the thigh should be undertaken with great caution in the average American hospital, because of the imperfect organization of many of these institutions. The short terms of unsalaried individual surgeons, the imperfect training and service of the usual resident physicians and nurses, and the dangers of prolonged anesthesia in unskilled hands make this method too risky when compared with the non-operative method. These faults in hospital organization militate somewhat against satisfactory results in non-operative cases also; but the danger to life is much less in the latter than in the former instance.

I report at this time two cases of death occurring after open

treatment and fixation by plates of fracture of the shaft of the femur below the small trochanter. I do this to show that such operative treatment is accompanied by risks that ought to be well considered by surgeons before deciding upon its adoption. In both of these cases I concluded to operate, because I believed it was likely that I could do better for the patient by rejecting non-operative measures. I thought I was giving the patient the best chance for a good leg and not especially imperilling his life. The result showed that I was mistaken.

I am quite sure that if all cases of death following the operative treatment of this important injury were recorded publicly, the medical profession would be convinced that there is a considerable risk accepted by a patient, who permits his surgical attendant to open a closed fracture of the femur and treat it by direct fixation.

TWO DEATHS FROM SUBTROCHANTERIC FRACTURE OF THE FEMUR TREATED BY DIRECT FIXATION.

Case I.—H. H., aged fifty years, who had a history of alcoholic addiction, though recently he had abstained from the use of alcohol to a great extent, on April 27, 1911, tripped over the carpet and fractured the left femur below the small trochanter. He was admitted to the Polyclinic Hospital on the same day and the site and character of the fracture were confirmed by an *x*-ray plate. The upper fragment was tilted upward by the action of the psoas and iliacus muscles. His temperature was about normal and the pulse about 80; respirations about 20. After treating the fracture for two days and finding it difficult to obtain apposition by Bucks extension and the inclined plane, I determined to open the soft tissues and to apply a metal plate to the fragments for fixation. (Fig. 1.) Accordingly, on April 29, I made an eight-inch incision on the anterolateral aspect of the left thigh and exposed the broken bone. By means of extension, applied with compound pulleys attached to the stirrup of the Buck's apparatus, the two fragments were brought into apposition and two rectangular metal plates adjusted, so as to fix the fragments in good position. One of the plates was put on the anterior surface of the bone, the other alongside of it, but more on the lateral aspect of the femur. The latter was twisted so as to give a hold around the shaft of the bone in a manner to prevent lateral as well as anteroposterior displacement. The wound was washed out with normal salt solution, the muscles approxi-

mated with interrupted catgut sutures, a gauze drain carried down to the site of the operation and the skin closed throughout the rest of the wound with silkworm-gut. A gypsum encasement splint was applied, the limb supported on an inclined plane, and Buck's extension by a few pounds used.

Within a few days the gauze was removed and the same line of treatment continued. Suppuration took place and the wound was then treated through a window in the gypsum splint; being irrigated with solutions of

FIG. 1.—Diagram of *x*-ray plate of fracture of the femur, which is difficult to keep in position by non-operative means. The fracture in Case I was similar to this diagram.

formaldehyde, and later with other antiseptics, for the purpose of averting septic processes in the wound. The temperature at the time the drain was taken out, which was about three days after operation, had risen to 101°. It fell to the normal and continued at about that point. Later the temperature rose, and an abscess was found in the wound. It was opened and the temperature curve again fell to about the normal line. In June, I transferred the patient to my colleague, Dr. Jopson, and asked him to remove the plates as I was about to go out of town. At that time there was a sinus at the point of drainage, from which pus was escaping. He

opened the wound, found the fragments in good position, and took out the plates; but the patient died on July 5, 1911, without having any special rise in temperature. No autopsy was obtained.

I believe Dr. Jopson considered the death to be due to exhaustion secondary to chronic alcoholism. During the entire time there were evidences of what I first looked upon as alcoholic delirium due to the patient's previous life. The man was restless at night, wandered in his conversation, but showed, if I recollect clearly, no albumin in his urine. Unfortunately the notes of the urinary examination were not preserved. Thinking that the solutions of formaldehyde used to irrigate the sinus and to dress the wound might be the cause of the mental aberration, I substituted other antiseptic washes. The man's condition did not seem to be modified by the change.

Case II.—J. D., aged sixty years, whose general health was good, although he had evidence of some hardening of the arteries, fell on the

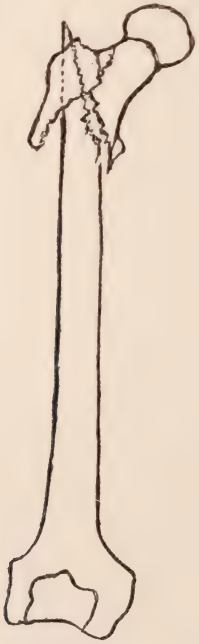

Fig. 2.—Diagram of *x*-ray plate of fracture in the case of J. D.

street, January 31, 1912. He struck on his right hip and fractured his right thigh bone a short distance below the smaller trochanter with the upper fragment split longitudinally upward. The skiagraph (Fig. 2)

shows an inverted V-shaped line of fracture running up into the neck. The patient's appetite was good, his bowels were regular, his thoracic organs seemed healthy on examination, he was alert in mind, and there was no albumin in his urine. He denied alcoholic addiction.

After endeavoring to keep the fragments in position by elevation on an inclined plane and Buck's traction method, I found that good apposition was not obtained, although I used 35 pounds weight upon the stirrup of the Buck apparatus. After giving the patient's condition careful consideration, and concluding that he was in even better health than I at first thought, I determined to make an incision and to try to reconstruct the bone by applying a Lane plate to the broken femur.

Through a long incision it was comparatively easy to apply a Lowman clamp, so as to hold the Lane plate and two fragments together. The shaft and the great trochanteric fragment were then fixed with the plate. Six screws were used. They were long enough to perforate the shell of the bone and pass through the marrow. They probably reached the opposite side of the bony cylinder. The other fragment of the upper end of the bone was so deeply buried in the muscles at the inside of the thigh that no attempt was made to fix it with screws or plate. It could probably have been done with a drill-fracture nail. Because of the great damage done to the soft parts by the original injury, I thought it wise to leave a gauze drain in the wound between two of the sutures. A wet dressing of liquor formaldehydi (1 to 500) was, I think, applied. Buck's traction apparatus was used, with a few pounds' weight, with the limb extended on the bed without the inclined plane, which had previously been employed. A gypsum encasement was not used because of my belief that there probably would be suppuration of the wound as a result of the extensive extravasation of blood.

This operation was done at the Polyclinic Hospital eight days after the injury. The patient's temperature after operation remained nearly normal for several days, varying from about 97° to 98½°. The pulse ran from about 80 to 100; the respirations from about 20 to 24. On the third or fourth day the wound was dressed and the gauze drain removed.

Nine days after operation I found that there was pus in the wound, though the temperature had not gone very high, being about 100° to 101°. On removing the stitches and separating the wound I evacuated about 4 fluidounces of pus. This occurrence induced me to introduce a rubber tube, which I carried from the lower part of the wound under the central stitches and brought out at the top of the wound. Irrigation was done daily with water and liquor formaldehydi (1 to 500) and the same strength of the antiseptic was used for an outside dressing. The strength of this solution was at times made weaker. Frequent dressing and covering the

wet gauze with waxed paper was necessary, because in using the urinal the man frequently saturated the bandages with urine.

About this time some impairment of resonance on the right side of the chest was noticed by me. Dr. David Riesman, my medical colleague, examined the man a few days later and confirmed the diagnosis of impairment of resonance of the right side, which he thought to be pulmonary and not pleural. He also thought there was some implication of the pleura on the left side. This condition was treated. Some time later the patient showed hebetude and had a very coated, dry, furred tongue. He presented an appearance very much like that of typhoid fever but without rise of temperature. His condition suggested to Dr. Riesman a catarrhal pneumonia, with pleurisy and perhaps a uremic state. The man then began to pass urine in the bed unconsciously and lapsed into a dull mental condition very different from his original alertness of mind, and died February 29, 1912

About the time that the wound showed so much infection he soiled the dressing on several occasions by spilling the contents of the urinal over his thigh. Urinary examinations were made quite frequently, and sometime after the operation a slight amount of albumin and a few casts were discovered. The patient also became delirious at times and rapidly lost flesh. Later the percussion note over the right lung seemed to be almost normal, and the left lung on ordinary examination appeared to be free from disease. There was very little rise in temperature during all this period, though occasionally it reached 101° or thereabouts. His pulse remained at about 80 or 100. His respirations while he had the more active lung symptoms went up to about 30 per minute, but they were generally about 20 to 24 per minute.

I had watched the wound carefully and found that there was no discharge of pus after the first few days, subsequent to the opening and draining of the wound, but the man's general condition was very unsatisfactory. I could detect no evidence of burrowing of pus and no active suppurative symptoms. I considered and discussed the propriety of opening the medullary cavity of the bone with a trephine during the latter part of his life, but was deterred from such operative treatment because the emaciated limb, the normal temperature, and the freedom from pain, swelling, or other evidences of suppuration in or around the bone convinced me that the wound was doing well.

My surgical colleagues, Dr. Morris Booth Miller and Dr. John H. Jopson, saw him and aided me with suggestions, but the man died on February 29, twenty-one days after operation. There was a slowly increasing failure of vital powers, with hebetude, increasing to coma, suggesting uremia or septic processes. The cause of the toxemia I could not explain to myself, although septic condition in the wound seemed more

probable than uremia or alcoholism alone. There was no color evidence of the blue pus of pyocyaneus infection observed.

No autopsy was allowed by his friends, but Dr. Rinker, of the resident medical staff, who removed the plate from the wound, reported the following local conditions: Two large pockets of pus were found, one extending up to the groin near the inner surface of the thigh; the other downward along the inner part of the thigh to about its middle. When the plate was removed the fragments separated and the marrow cavity of the upper fragment contained bloody purulent material. The soft tissues of the incision had united, with the exception of the drainage opening, but the muscles and interstitial tissues of the inner aspect of the thigh were in a necrotic state. The contents of the two pus pockets did not look like pure pus, the fluid was more viscid than true pus. It was of a dark reddish color and contained the debris of necrotic tissue. The capacity of the upper pus pocket was about five fluidounces.

Study of these two cases shows, I think, that the latter died of septicemia arising from purulent inflammation in the bone-marrow and in the tissues around the bone; and that the former probably succumbed from the same cause, though chronic alcoholism may have been a factor in that death. The symptoms were very similar, except that the first man died about three weeks after operation, the second lived nine weeks. Both were laboring men beyond middle life, and therefore perhaps injudicious selections for the so-called "bloody" treatment of femoral fracture.

I, however, decided to operate upon each of them instead of continuing non-operative methods, because I realized that a laboring man finds a deformity or an incapacity from fracture of the femur a greater handicap to his career than would a patient belonging to a higher social group. Both stood the shock of operation well. I watched both carefully; one until death, the other until, shortly before death, I transferred him to the care of a well-known skilled surgeon. Nevertheless, death came to both, because I fastened the displaced fragments together by direct operation, thereby converting closed fractures into open ones. Perhaps vertical traction, such as we use in fracture of the femur in children, or the Smith or the Hodgen anterior wire splint would have given a good apposition of fragments.

It may, however, be said that these men might have died under non-operative treatment. Such instances are known to most surgeons. While I had the second case under my care a woman of about fifty years was admitted to my ward with a badly comminuted fracture of the cervical and trochanteric region of the left femur. I was unable to keep the fragments in proper apposition by various external fracture dressings which I tried, and then

Fig. 3.—Diagram of *x*-ray plate of patient who refused to have fracture through neck of femur nailed.

advised her to let me drive a nail or screw through the large trochanter for fixation. (Fig. 3.) She refused. Non-operative treatment was therefore continued. She became stuporous and delirious, removed her dressing, and had unconscious urination. As a result she developed bed-sores and presented almost identical symptoms with those of the two men, whose death I report. She is now slowly recovering.

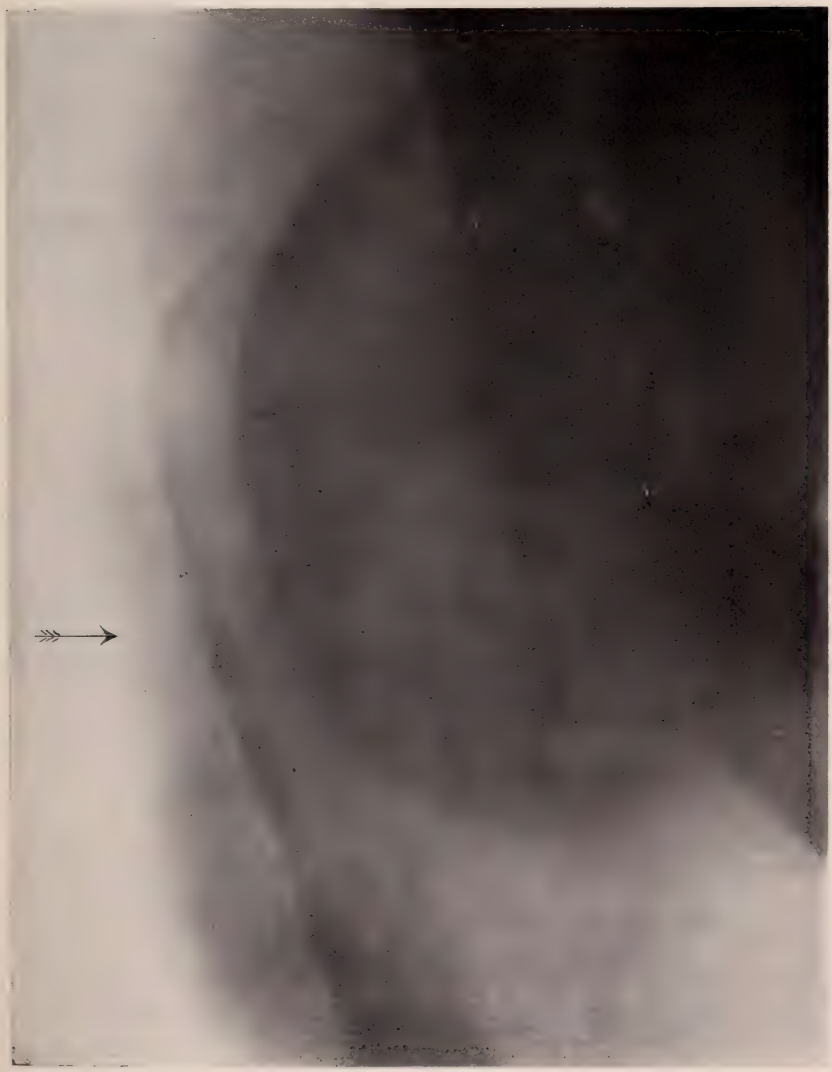

FIG. 4.—Dr. Roberts' patient, a woman, operated upon February 26, 1908. Great anterior deformity, due to malunion of fracture below small trochanter, caused by flexion and outward rotation from action of iliopsoas muscle. Resection of the ends of fragments and fixation with Roberts' fracture nails. Cured with good limb. Illustration shows the manner in which *x*-ray plates are deceptive unless taken in two planes or stereoscopically. Here there is scarcely any appearance of fracture or of displacement shown.

During the same period of time one of my patients, who had received a bad spiral fracture of the left femoral shaft, while suffering from a gonorrheal discharge and cystitis, unexpectedly died, within a day or two, with pulmonary symptoms, although he had been convalescing satisfactorily for a couple of weeks. The man's improvement in the initial symptoms of the fracture, the decrease in the discoloration of his thigh, which had been caused by the submuscular and subcutaneous extravasation of blood, my frequent examinations and his general aspect of doing well gave no

FIG. 5.—Staples applied to comminuted fracture of tibia. The legs of staples were too thick and did not go down in drill holes far enough to bring staples close to surface of bone. Dr. Roberts' patient, P. G. Operation, April, 1905.

warning. Unfortunately no autopsy was obtained, but clinically the death seemed due to pneumonia from influenza infection. Fat embolism did not seem likely.

It is also proper for me to recollect that operative attack on the femur may be followed by fatal surgical shock. A dozen or more years ago I had a young girl of sixteen or eighteen years of age die a few hours after excision for ununited fracture of the shaft of the right femur. She was in perfect general health and had previously been operated upon in another city for non-union of the

Fig. 6.—Result in P. G.'s case of comminuted fracture of tibia and fibula five months later.

FIG. 7.—Vicious union, right humerus. Treated by osteotomy and nailing with fracture nail in March, 1901. Wire attached to head of nail and brought out of wound to facilitate search for nail when it was to be removed. Nail removed at five weeks. Dr. Roberts' case.

broken bone. That operation had failed to induce union. She was very much frightened when she came to the operating room and I myself started the anesthesia. I resected the ends of the fragments so as to make a mortise and used either screws or wire to hold the bone ends in apposition. The procedure was rather long and difficult, but she did not lose too great an amount of blood and was not badly etherized. Still, while on the stretcher on the way to the ward she showed suddenly signs of collapse and died in a few hours despite my best efforts to cause reaction. I think that she was carried up stairs head first, which may have caused injurious cerebral anemia. As is so unfortunately and so commonly the case in our American surgical work, no autopsy was obtained to determine the psychic or anatomical cause of death.

As I have already said, a purpose in reporting these deaths, after the now fashionable operative treatment of fractures by direct fixation, is to neutralize, if possible, the effect of the unwise enthusiasm of some surgical writers.

I admit that in my cases I may have been mistaken in employing drainage for a few days; the perforation of the marrow cavity of the bone may have invited unnecessary medullary sepsis; the dressings of formaldehyde, after the wounds became infected, may have given rise to toxic symptoms through absorption. Still, the fact remains that death took place in two patients under the care of a surgeon who thought, perhaps presumptuously, that he had sufficient experience to undertake the work and to decide on its advisability.

For years I have advocated the open treatment of broken bones in selected cases of closed fracture, and have occasionally resorted to the method (see Figs. 4, 5, 6 and 7). This recent experience has nevertheless proved to me that our profession must be guarded in its advice relative to the general adoption of operative methods in the management of fractures. Some of these injuries demand operative treatment, some will permit its employ in competent hands and under proper surroundings; but others will be followed by unnecessary fatalities. In the last group, some of the patients will have a better chance for life, if less strenuous methods are used by the surgical attendant.

This paper is a plea, if I may use that term, for a conservative progressiveness in the surgical art. Enthusiastic adoption of the newest suggestion may lead to disaster, if not restrained by conscientious consideration, careful observation, and logical thinking. I have no words but condemnation or pity for the obstinate, unseeing, and unhearing "holdback" in science; but unthinking, rash, and dogmatic statements without the straight thinking, recently so well advised by a well-known college professor, are unscientific and cause in the minds of many of us a psychic emesis. They may even lead some hearers to become too conservative, and reject the seeds of truth, and thus do harm. They certainly in surgery will, as they have heretofore, fill some untimely graves.

DISCUSSION

Dr. G. G. Davis: Some years ago before the appearance of Dr. Lane's paper in advocacy of plates, I had a patient with fracture at the juncture of the upper and middle third of the femur upon whom I operated, wiring the bone. The wound healed and one might almost quote the saying that "the operation was a success, but the patient died." The difficulty was that, notwithstanding the prompt healing of the wound, before the femur was sound enough to allow the patient to get out of bed, the difficulties in taking care of him were such that he died from bed-sores due to failure of the nursing. I think Dr. Roberts is right in calling attention to the severity of operations on the femur. Personally, I think that operation on this bone is often very difficult, whether for recent, ununited, or various other fractures. Hemorrhage is often very free and comes from the worst possible point, deep, posteriorly, from the perforated arteries which come down the linea aspera. I think the operation is sufficiently difficult and dangerous to cause a comparatively inexperienced person to hesitate.

Dr. Richard H. Harte: I think we owe Dr. Roberts a vote of thanks for bringing this question before us, and particularly for his frankness in regard to the results in the two cases which he has reported.

There is a feeling now, on the part of some of the more radical members of the profession, that the only rational way to deal with fractures of the thigh is by the method pursued by Mr. Lane, namely, of opening

the thigh, exposing the ends of the bones, adjusting the fragments, and retaining them in position with screws and plates.

Before this more radical method was advised, the older surgeons obtained fairly good results, and the reason they obtained them is that they devoted much more time and attention to looking after the fractures than at the present time, when there is so much to occupy the mind of the operating surgeon. It is now only after a case is noted to be doing badly that the attention of the attending surgeon is called to it, and then there is little left to do but to operate.

My feeling is that the cases to be operated upon are those which we find are not going to unite, or if so, will unite with a great deal of deformity. In cases where this has been determined by carefully made skiagraphs, radical interference should be resorted to, because we invariably find a piece of fascia or fragment of muscle separating the ends of the bones, so that osseous union is impossible.

If a case should not promise to be a good surgical risk, it is much wiser not to operate, and the patient had much better submit to an ununited fracture, or even vicious union, than to succumb to the complications attending operative procedure. I think it is a good working rule to wait at least two weeks between the receipt of the injury and interference, in simple fractures, because, in that time, nature has thrown out certain barriers which make these cases less liable to infection than they are after a recently fractured bone, where considerable hemorrhage has taken place and all the conditions are favorable for infection.

These cases, at best, are difficult to deal with and must be treated by a man who has a great deal of surgical skill and ability and all the necessary appliances for handling this rather complicated class of cases.

Dr. John H. Jopson: I saw both of these patients of Dr. Roberts, one after the operation, and the second one at the time of operation, also subsequently. I think a word is due regarding Dr. Roberts' well-known courage and honesty in reporting these cases. The first patient I think died of alcoholism, and as he died under my care after Dr. Roberts had left the city I had opportunity to convince myself of this. Of course, the alcoholic symptoms were aggravated by the accident and the operation, and there was here a low grade of sepsis in an old man confined to his bed, and often patients die under such conditions when no operation has been performed. The second case was exceptionally difficult, and the fragments required much handling at operation, which favored the infection which ended fatally. There has been a tremendous wave of enthusiasm in regard to the open treatment of fractures, and I think we all agree that the report of these cases at this time is very timely. There is no doubt about the fact that the open treatment of fractures has come to stay and the indications for operation will be more and more broadened

with growth in our knowledge and experience; but we must check up our results. It is essential that we equip ourselves with those appliances which Dr. Harte mentioned and which Dr. Martin has helped to devise, as well as qualify ourselves with surgical experience and judgment. We all know that much operative work is done by general practitioners who want to break into surgery. In a field where the opportunities for operation are so abundant and the literature teeming with suggestions of new methods we must take special care to point out the dangers present.

Dr. Edward Martin: It is at Dr. Roberts' request that I discuss this paper. I explain this because the hour is a little late and the program is somewhat long. Dr. Roberts brings up many points for careful consideration, but the dominant note in his communication is that of altruism, which leads him to report unsuccessful results. He reports two fatalities following operation on fracture of the upper extremity of the femur in people past middle age. Even the conservative treatment gives an astonishing mortality in these fractures, something over 10 per cent.; they die of a form of toxemia characterized by anorexia, general feebleness, and delirium. The usual treatment is to get them out of bed. This is usually as futile as is keeping them on their backs. The best treatment we have used has been the application of plaster of Paris with the reduction under anesthesia. In one case, characterized by delirium, anorexia, fecal and urinary incontinence, and progressive asthenia, in twenty-four hours there was an entire change in symptomatology, the patient promptly recovering.

I have one case to add to Dr. Roberts' list—a case of double fracture of the femur, with a shortening of two inches. Extension was tried for over two weeks, with unsatisfactory result. Open operation and reduction was done by skilled men, but the man died of shock. We had one or two cases of active suppuration which got well; one or two of very slow suppuration. I feel convinced that every time a foreign body is put into a fracture, union is delayed. Absolute apposition will not insure the union of a fracture. My own feeling is not to operate unless operation is imperative. The ease with which the operation can be done is in inverse proportion to the time elapsing after injury. An operation in twelve or fourteen months after the fracture is extremely difficult.

In making traction on the bone we have added the principle of taking blood pressure and watching the pulse. Upon the rapid reduction of the blood pressure traction is stopped and section and plating are rapidly completed. In old fracture of the femur with the point of fracture deep and hard to get at requiring mechanical force, infection is not infrequent. I feel that Dr. Roberts is perfectly correct in saying that if the true history of operative treatment of fracture of the femur were known the practitioners would be very slow in referring their cases for operation.

Dr. William L. Rodman: I think Dr. Roberts is to be commended and congratulated for this paper; commended for its candor; congratulated for its timeliness. As Dr. Martin has just said, there are probably a great many unreported cases throughout this and other cities on account of this wave of enthusiasm for the open treatment of fractures. Dr. Roberts has told us plainly that there must be good reasons why an operation is done on a certain fracture—non-union, vicious union, or other good reason why a good result cannot be obtained without open treatment. Again he pertinently says, that you must do more for a laboring man than for a capitalist to whom complete usefulness of the limb is not so essential. I think operation is the treatment for the exceptional case, and, as Dr. Roberts has said, the operation is one calling for an unusually skilled and careful surgeon. I believe that fractures of the patella perhaps are nearly always best operated upon, because here the functional results are not good without operation in the vast majority of cases. Hence I think the open method is best, and although I lost my first case of operation for fracture of the patella in 1896, I have operated in this way on all patients aged under fifty years that I have seen since, and all of these have done well. I have operated upon only one case of fracture of the femur, and that recovered nicely; but have operated upon a number of clavicles, humeri, and tibiæ particularly. Only a few days ago a fracture of the femur was sent to me in a bad position, the lower fragment overlapping the upper fragment; I was very unwilling to break up this vicious union, for I saw at once that there might be fatal hemorrhage because of the proximity of the popliteal vessels. Under anesthesia I broke up many adhesions, and was able to get a good result. The patient was sent home much improved. I agree with Dr. Harte that it is not best to operate within the first twenty-four hours or within the first week, but that we should wait until there is absorption of the effused products. It is particularly my rule in fracture of the patella to operate during the second week rather than the first.

Dr. Roberts (closing): I have come to the conclusion in regard to these subtrochanteric fractures that a better way than the open operation is probably to put my patient on a level and firm bed, with the thigh in the vertical position, and apply traction with a weight and pulley to overcome the deformity, just as is often done in young children.

TUBERCULOUS MENINGITIS: A PATHOLOGICAL REPORT OF NINE CASES OF MENINGITIS[1]

By JOHN H. W. RHEIN, M.D.

I RECENTLY had the opportunity of examining histologically 9 cases of meningitis, 7 (Cases I, II, III, IV, V, VI, VII) of which had been diagnosticated clinically and pathologically as tuberculous meningitis. Case VIII had been diagnosticated as one of tuberculous meningitis from which the patient recovered only to succumb a month later during an attack of pneumonia. The findings are interesting as showing the histological features of a recovered case of meningitis.

Case IX was diagnosticated clinically and pathologically as pneumococcic meningitis and is of interest in comparison with the findings in the tuberculous cases.

As far as possible the material was taken from those regions of the cortex where the tubercles were not found. While all the sections were stained for the tubercle bacillus, none was discovered in any case after careful search. It should be mentioned that some of the specimens were hardened in Kaiserling, which may have affected its staining properites for the tubercle bacillus. The results were interesting and instructive, and though they do not permit the drawing of any positive conclusions, they suggested some ideas which are worthy of reporting.

It seemed possible that the lesion, which was fairly uniform in its histological character, was not alone caused by the tubercle bacillus, for reasons which will be developed later on in this paper.

[1] Read May 1, 1912.

It further seemed possible that the meningitis was due to a mixed infection. If this could be proved it would seem but a step further to obtain a vaccine which might operate against such a process. If this could be successfully combated, there then would remain the tuberculous process alone, which in itself may be looked upon as less fatal than the mixed infection, and within the possibility of cure.

Experiments are now on foot in conjunction with Dr. Herbert Fox with this end in view, and a report will be made later embodying the results of these experimentations.

I am indebted to Dr. J. P. Crozer Griffith, Dr. J. C. Gittings, and Dr. Alfred Hand for the privilege of reporting some of these cases, and to Dr. C. Y. White for the pathological material from the cases from the Children's Hospital.

Case I.—A. P., aged twelve years, was admitted to the Children's Hospital September 14, 1905. The family and previous histories were negative. The symptoms dated from the day before admission and consisted of fever, delirium, persistent vomiting, headache, drowsiness, and slight stiffness of the neck. On admission the abdomen was distended and there was obstinate constipation. Examination of the lungs was negative and also of the heart except that it was rapid. The pupils were dilated and equal. Kernig's sign was present. The head was retracted and tâche cérébrale was demonstrated. Numerous dark brown spots scattered over the body were seen. Later the legs became rigid, the face was drawn to the right, and deafness ensued.

Examination of the eyes by Dr. Wm. Campbell Posey was negative, except that the pupils were dilated.

An irregular temperature marked the course of the disease, ranging from subnormal to 104.6°. The patient was most of the time in a semi-stuporous state. The child died November 26, 1905.

At the autopsy the dura was opaque and thickened. The pia-arachnoid was also opaque and thickened, and areas of exudate were observed on the base, the convexity, and along the Sylvian fissure. A few miliary tubercles were observed. No tuberculous process was seen elsewhere.

Microscopic Study. The pia was the seat of a round-celled infiltration, the cells being mainly of connective-tissue type, though there were some mononuclear round cells. Connective-tissue formation enclosing blood pigment and numerous blood cells was also observed. The connective-tissue cells showed in places vacuolization, and some contained blood pigment.

The intima of the arteries and veins was thickened and showed proliferation of the connective-tissue cells.

The pia of the medulla was the seat of a marked round-celled infiltration, the cells consisting of small mononuclear cells and large polynuclear cells.

In the cortex there was a marked degree of perivascular dilatation, but the cortical cells stained well.

This case was of the mixed type, the proliferative type predominating in the pia of the cerebral cortex, and the exudative type in the pia of the medulla. This case was clinically one of tuberculous meningitis, and while no tubercle bacillus was found in this or, indeed, in any of the cases of this series, microscopically it coincided with the usual histological characteristics of this disease.

Case II.—M. H., aged thirteen months, was admitted to the Children's Hospital March 16, 1906. Family and previous histories were negative. The patient died March 25, 1906, seven weeks after the onset of the disease, though the nervous symptoms preceded death but three weeks.

The first symptom consisted of diarrhea with green stools. A month later the child was seized with convulsions affecting the left side of the face, arm, and leg. The eyes were turned toward the right and later the right side of the face, the right hand, and the foot were convulsed. The movements were more or less continuous and there was some general rigidity which later became very marked, the legs and arms being held in extension. Kernig's sign was not clearly demonstrated. The knee-jerks were not increased. Lumbar puncture was done without effect upon the symptoms. The heart was rapid and there was some dyspnea. There were no spots on the body and examination in other respects was negative. The temperature ranged from 99.2° to 104.4°.

At the autopsy there was found general tuberculosis involving the lungs, epicardium, peritoneum, kidneys, stomach, intestines, and lymph glands.

Microscopic Examination. The infiltration of the pia consisted mainly of cells of the connective-tissue type. Many plasma cells, however, were present. Numerous red blood cells were found in the thickened pia. The veins were slightly thickened, the intima showing a marked proliferation of the connective-tissue cells. There was a slight perivascular distention and slight perivascular infiltration of the cortex.

In this case in which the nervous symptoms manifested themselves for three weeks only before death, there is evidence clinically of an acute process. The type of cellular infiltration is strikingly proliferative. The presence of blood in the process is worthy of mention as being present in both the exudative and proliferative forms of tuberculous meningitis.

Case III.—R. S., aged six and a half years, was admitted November 14, 1906, to the Children's Hospital and died November 28, 1906. The

family and previous histories were negative. Duration of the disease was twenty-two days, and began with loss of appetite and headache and slight enlargement of the tonsils and fever. Upon admission the examination of the lungs and heart, abdomen, and spleen was negative. There was marked tâche cérébrale. The patient was irritable and lay curled up on one side. There developed then rigidity of the neck, dysphagia, and general convulsions. Kernig's sign was present on the sixth day. The patellar reflexes were irregular, being sluggish on the right. Paralysis of the left external rectus was present, associated with nystagmus. Babinski phenomenon was present and finally Cheyne-Stokes respiration.

At the autopsy a diagnosis of tuberculous meningitis was made, though no note of the presence of any tubercles was made at the time of the necropsy.

Microscopic Examination. The pia showed the presence of a dense infiltration which contained many mononuclear and polynuclear leukocytes. In other places the cellular infiltration consisted largely of endothelial cells which was very marked around the bloodvessels. Red blood cells were numerous and a number of plasma cells were observed. Where the tissue was loose the cellular infiltration was composed largely of lymphocytes and leukocytes, but where the tissue was denser the connective tissue cells predominated. The bloodvessels showed some thickening of the walls, and in places accumulation of round cells beneath the intima was pronounced.

In the cortex there were perivascular distention and slight round-celled infiltration around the bloodvessels. Marked encephalitis was observed, the leukocytes predominating in the exudate.

This case illustrates the mixed form, showing the presence of both the exudative and proliferative type of tuberculous meningitis.

Case IV.—M. E., aged nineteen months, was admitted to the Children's Hospital April 24, 1906, and died two days after admission. Her mother had died of tuberculosis four months previously, but otherwise the family history was negative. The patient had suffered from a cough for four months previously and general irritability.

Two days before admission she became first dull then unconscious. She was able to move the left side of the face only and the eyes twitched at times. There was general rigidity, more on the right side. The left eyeball was drawn inward and there was also palsy of right external rectus. Kernig's sign was not definite.

In the lungs coarse moist and fine dry rales were audible. The spleen was palpable and the liver enlarged. Examination of the heart was negative. The temperature ranged from 98° to 102.8°.

At the autopsy a diagnosis of general tuberculosis was made, involving the lungs, liver, spleen, kidneys, intestines, and meninges. In the

meninges the exudate occupied the posterior and superior surface of the cortex posterior to the middle line and the base of the brain.

Microscopic Examination. In the pia the infiltration was made up of leukocytes and lymphocytes mainly. In places there was present in the pia large quantities of red blood cells. The process was mainly of an exudative type. The cortex was invaded in places, the cells in the subcortical layers being of the mononuclear type and containing darkly staining nuclei, but polynuclear cells and some plasma cells were also observed. Perivascular distention of the cortex and round-celled infiltration around the vessels were present. Obliteration of the small vessels with some slight proliferation of the intima in others was noted.

This is an example of the purely exudate type of meningitis. The case was a rapid one, the evidence of meningeal implication lasting but four days.

Case V.—H. P., aged two years, was admitted to the Children's Hospital March 25, 1906, and died May 31, 1906. The family and previous histories were negative except that one sister died in spasms. There was no history of tuberculosis.

The child had been sick for two weeks before admission, making the duration of the disease eighty-one days. The disease was ushered in with vomiting, constipation, slight cough, and somnolence.

Upon admission the child was unconscious; the eyes were open, the pupils large, reacting sluggishly, strabismus was present, and there was rigidity of the neck and extremities. The knee-jerks could not be developed, though ankle clonus and the Babinski phenomenon were present. Kernig's sign was positive. The temperature ranged from 98° to 100° until just before death, when it rose to 105.2°.

At the autopsy a diagnosis of tuberculous meningitis was made and miliary tuberculosis of the lungs, liver, kidneys, spleen, intestines, and bronchial glands was present. There was an exudate over the motor region on the right side and also at the base.

Microscopic Study. The pia was thickened, this being due to fibrous tissue containing many connective-tissue cells and a few round cells. Some red blood cells were found in the thickened pia. The veins were surrounded by many connective-tissue nuclei, but the bloodvessels themselves were not implicated.

The cortex was invaded by the process. There was some perivascular dilatation and the bloodvessels were distended and showed slight proliferation of the intima.

The proliferative type of tuberculous meningitis predominated in this case. The process was not extensive nor very intense, and was evidently, as the clinical story would indicate, a chronic process.

Case VI.—B. P., aged three and one-half years, was admitted to the Howard Hospital January 12, 1908, and died January 13, 1908.

The family and previous histories were not obtainable.

Patient on admission showed paralysis of the right side of the face, right arm, and leg. The right arm was flexed and was distinctly spastic. The left arm and leg were kept in constant motion. Movements of the head were painful and disclosed stiffness of the neck muscles. The right biceps-jerk was increased, but the left could not be elicited. Both knee-jerks were increased and equally so. Babinski phenomenon was present on both sides, but ankle-clonus was absent. The pupils reacted normally. Temperature ranged from 102° to 105°.

The autopsy revealed the presence of tuberculous meningitis and hydrocephalus. Miliary tuberculosis of the kidney and peritoneum was observed.

Microscopic Study. The pia from the paracentral region was the seat of a dense process which implicated both the pia and the superficial cortical layers. Connective-tissue cells predominated, though there were some trifoil cells, but these were mainly perivascular and were present where the infiltrative lesion was less dense. The connective-tissue cells were found in various states of degeneration, many of them showing vacuolization. In the superficial layers of the diseased pia the loose connective tissue was infiltrated with mononuclear and polynuclear cells, and a few plasma cells, and was also the seat of extensive extravasation of blood.

In the frontal region the same condition obtained. In this region the bloodvessels were diseased, the outer walls being the seat of cellular accumulations. The cortex had been invaded slightly in places, the tissue being loose and the seat of some space formation. Just below the pia there was present cellular infiltration, some cells being mononuclear round cells, some being pale and granular without a demonstrable nucleus, some elongated cells, some cells with two nuclei, some trifoil cells, and some large cells with many nuclear fragments tending to undergo vacuolization. Phagocytosis was also observed in the subcortical layers.

The cortex of the cerebellum was the seat of a marked encephalitis. The pia of the cord was the seat of a mononuclear round-celled infiltration of moderate intensity and the posterior roots were infiltrated with cells of a similar nature. The anterior roots were similarly affected, but to a less degree.

This case belonged to the proliferative type of tuberculous meningitis, but the characteristics of the exudate type of the disease was also present to a marked extent, and it therefore more properly comes under the head of the mixed type.

Case VII.—V. S., aged two years, was admitted to the Howard Hospital November 4, 1907, and died November 8, 1907. The family and previous histories were unobtainable. The child was admitted with tumor of the abdomen, which proved to be a distended bladder.

The patient was semicomatose on admission. The pupils did not react to light and the eyeballs were moved from side to side constantly, there being a tendency to left conjugate deviation, with transient weakness of the left internal rectus. The scalp and the nape of the neck was tender to pressure and the head was retracted. There was general rigidity of the limbs and from time to time a chronic spasm of both arms. Tâche cérébrale was present. The knee-jerks could not be elicited, probably due to the rigidity, and the Babinski phenomenon was observed on both sides. The examination of the eye-grounds by Dr. Wm. Campbell Posey was negative.

At the autopsy general tuberculosis was found of the lungs, liver, mesenteric glands, and also the meninges, though macroscopically no tubercles could be observed in the process in the meninges.

Microscopic Study. The pia was much thickened and contained leukocytes and connective-tissue cells. The cells were mainly of the connective-tissue type, though many plasma cells were present and some red blood corpuscles. The bloodvessels showed marked cellular proliferation of the walls. Around the bloodvessels there was marked cellular accumulation of connective-tissue cells.

The cortex was the seat of encephalitis in places, and there was perivascular distention, the cortex being in places rarefied.

This case belongs to the proliferative type of tuberculous meningitis.

Case VIII.—J. R., aged six years, was admitted to the Children's Hospital January 27, 1906, and died January 30, 1906. The family and previous histories were entirely negative. She had previously been treated at the Children's Hospital for an attack of meningitis, which lasted from December 1, 1905 to December 28, 1905, when she was discharged cured, save for paralysis of the external rectus muscle of the left eye.

At that time she had suffered with rigidity of the cervical muscles and of the legs which were held in extension. Kernig's sign was present and there was paralysis of the external rectus of the left eye which remained paralyzed. The knee-jerks were absent and there was no ankle clonus or Babinski phenomenon present. The pulse was irregular and intermittent. The temperature was not characteristic and ranged from 97° to 104°.

Examination of the fluid obtained by lumbar puncture showed the presence of intercellular diplococcus and an excess of polymorphonuclear cells.

A month after her discharge she returned with symptoms of lobar pneumonia from which she died in a few days.

Pathological Diagnosis. Double lobar pneumonia, acute fibrinous pleurisy. The lung showed some cavity formation, and pulmonary tuberculosis including some miliary tubercles, was probably present. The arachnoid was congested except over the frontal lobes.

Microscopic Examination. The infiltration in the pia was not intensely cellular. There were some leukocytes in small quantities, numerous round cells, numerous connective-tissue cells, and some large round mononuclear cells. Many red blood cells were seen. Some of the connective-tissue cells showed advanced vacuolization and some contained blood pigment. Other cells stained deeply and contained dark round nuclei.

The vessels of the cortex were filled with red blood cells, the walls were thickened, showing hyaline degeneration, and some of the vessels were obliterated. There was perivascular round-celled infiltration of the cortex which was rarified. Fresh hemorrhages were found in the subcortical layers which was also the seat of a slight cellular infiltration.

In this case a diagnosis of tuberculous meningitis had been made from which the patient made a recovery, having been discharged cured. It may have been and probably was a diplococcus meningitis, though the presence of cavity formation in the lungs leaves some doubt as to whether or not it was not actually a case of tuberculous meningitis after all. This case is interesting as illustrating the alleged cured cases of tuberculous meningitis, about the exact nature of which, however, it seems to me there is some doubt.

The microscopic findings were similar to those found in tuberculous meningitis of a proliferative type, but less marked. Added to this there was evidence of an old process in the fibrous nature of the process and in the presence of degenerated cells.

The cortical lesion was of two kinds—an old process, represented by the rarefication of the cortex and degenerated bloodvessels, and a recent process characterized by fresh hemorrhages and some round-celled infiltration, perivascular in type.

The older process, I take it, represented what remained of the diseased meninges from which the child recovered apparently a month prior to the onset of the fatal pneumonia. The more recent process could be explained by a meningeal extension of the pneumococcic invasion.

Case IX.—J. S., aged one year, admitted to the Children's Hospital September 26, 1907, died October 21, 1907. The family and previous history were entirely negative. The child was breast-fed from birth. The duration of illness was thirty days and began with high fever and vomiting. Shortly after the onset the patient was comatose, the neck became stiff, the pupils were dilated, reacting sluggishly to light, and the reflexes became increased. Except for two vesicles which were found upon the cheeks, no eruption was observed.

Later there developed internal strabismus, tremor of the head and body, spasm of the hamstring muscles, and Cheyne-Stokes breathing.

Lumbar puncture was made on several occasions and contained besides

pneumococci many polynuclear cells, leukocytes, and red blood cells. The temperature ranged from 98.2° to 105°, rising finally to 107.2° before death.

At the autopsy cerebrospinal meningitis was diagnosticated. None of the other organs showed any tuberculous involvement. There was marked distention of the fourth ventricle.

Microscopic Examination. The pia was fibrous and sparsely dotted with cells, mononuclear in type. The nuclei stained poorly in the outer layers of the thickened pia and in some the protoplasm was granular while in others there were two nuclei present. In the inner layers there were many deeply staining, small, and irregular nuclei. A few plasma cells were observed. Around the bloodvessels the cellular accumulation was marked, composed mainly of the connective-tissue type of cell.

There was also a slight proliferation of the intima of the small veins.

In the cortex capillary hemorrhages were observed, and there was some perivascular distention. In some of the cells four nuclei were observed, suggesting karyokinesis.

This case was diagnosticated as pneumococcus meningitis, the pneumococcus having been found in the cerebrospinal fluid. It is of the mixed type, showing proliferative and exudative characteristics, and it is to be noted that microscopically it differed in no way from the usual picture of tuberculous meningitis.

Summary. Of these cases only 1 (Case IV) properly belongs to the purely exudative type of tuberculous meningitis; 3 (Cases II, V, VII) are proliferative cases and 3 (Cases I, III, VI) are of the mixed type. Blood or blood pigment was found in 8 (Cases I, II, III, IV, V, VI, VII, VIII) of the cases. Plasma cells were found in 6 of the cases (Cases II, III, IV, VI, VII, IX).

Bloodvessel changes to a more or less degree were present in all the cases. Cortical changes were present in all the cases, in three of which marked evidence of encephalitis was observed. The duration of the disease seems to have little if any bearing on the character of the exudate. The duration of the disease in the exudative type was four days; in the proliferative type three weeks, twenty-seven days, eighty-one days, and one of short duration; in the mixed type seventy-four days; thirty days, twenty-two days, and one of short duration.

In this study an effort was made to investigate the histological characteristics of the process at those places removed from the

tubercles themselves. The lesion in all cases was more or less similar except in the one case showing a purely exudative type and this was one beyond doubt of typical tuberculous meningitis.

Cases VIII and IX are doubtful cases of tuberculous meningitis but the histological findings are identical to those found in the other cases.

This brings up the question as to the nature of so-called tuberculous meningitis. I believe it is possible if not probable that the meningeal process in these cases is due to a mixed infection and not alone or at all to the toxins of the tubercle bacillus.

The rapidity of the course of the disease in these cases, which from its inception until the death of the patient is often but a very few days, is unlike the slow chronic process characteristic of tuberculosis in other organs. Moreover, in cases of tuberculosis of the meninges of cases which succumb to this disease in other organs, the histological picture is not the usual one found in the tuberculous meningitis of children.

The process in tuberculous meningitis, I believe, does not necessarily originate solely from the tubercle bacillus or its toxins. The absence of the bacillus of Koch in the exudate which is not unusual, speaks against it. Moreover, the histological picture of the lesion removed from the tubercle is of a round-celled infiltration, which, I believe, cannot be said to be pathognomonic of a tuberculous process.

Non-specific or simple inflammatory reactions in tuberculous meningitis have been already described by Villaret and Tixier,[1] Peron,[2] Hayen,[3] Chantemesse,[4] and others.

Diffuse leukocytic infiltration without tuberculous granulations and without definite tubercles, and giant cells have also been observed by Siredey and Tinel.[5]

That it may be a mixed infection is well illustrated in the observations of Perrin,[6] who found the diplococci in the cerebrospinal fluid; of Kneass, Hendrickson, and Sailer,[7] who isolated the micrococci tetragenous in the spinal fluid as well as the tubercle bacilli in the smears from the tubercles in the meninges; of Griffon and Abrami,[8] in whose case the cerebrospinal fluid was invaded by the

coli bacillus; of Marotte,[9] who described a case of septicemia of tetragenous origin coincident with tuberculous meningitis; and of Poisseau and Tixier,[10] in which were found diplococci not taking the Gram stain.

In two cases in my series, moreover, the diplococcus was found in the cerebrospinal fluid. In this connection, however, should be mentioned the contention of Sicard[11] that the exudate in the pia is not the result of polymicrobic infection, and that the bacillus of Koch and its toxins are capable in themselves of creating these granulations.

The experimental evidence is somewhat conflicting. Hektoen[12] failed to produce typical meningitis by injecting tubercle bacilli into the carotids of rabbits though miliary tubercle were observed in the meninges.

On the other hand, Sicard experimentally produced a diffuse process consisting of a leukocytic infiltration and other lesions similar to the diffuse meningeal changes found in man. There is no evidence in these experiments to exclude, however, the existence of a mixed infection.

At the same time the experiments of Martin and Vandremer,[13] and Peron[14] show, according to these observers, that the toxins of tuberculosis play an important role in the development of tuberculous meningitis.

Cellular Changes. The cellular changes have been studied by Diamond,[15] Josue and Salomon,[16] Poisseau and Tixier, Higgs[17] and Siredey and Tinel.

Diamond found that the plasma cells, lymphoid cells, and phagocytic cells formed the principal cell content of the infiltration of vascular leptomeningitis. He claims to have been the first to have described the presence of the plasma cell in acute tuberculous inflammation. The phagocytes were looked upon as transmitting the tubercles from one area to another.

Lymphocytes and polynuclear cells predominated in the cases of tuberculous meningitis in adults studied by Josue and Salomon.

In the case cited by Poisseau and Tixier the cellular infiltration was composed of mononuclear lymphocytes and endothelial cells

without giant cells, and finally in the case of Siredey and Tinel the examination showed a diffuse leukocytic infiltration without tuberculous granulations or giant cells.

The exudate in Higgs' case, consisted of fibrin and round cells, mainly large and small lymphocytes, but some polymorphonuclear leukocytes.

The cellular changes of the exudate of tubercular meningitis are not absolutely characteristic, somewhat similar changes having been described by Spielmeyer,[31] as existing in trypanosomiasis and paresis (Gehry), though Gehry concludes that the miliary foci in the pia give the process a typical appearance. While this is true, I think it still may be contended that the cellular makeup of the process remote from the tubercle is not typical of a tuberculous process.

Vascular Changes. The bloodvessels exhibited, in my cases, more or less change in 7 of the 9 cases studied. These changes consisted of thickening of the walls of the arterioles and some veins, which in one case went on to obliteration of the lumen of the small vessels. Perivascular round-celled infiltration was observed quite commonly. In some instances the coats of the veins were thickened and in others there was cellular proliferation of the intima. In one case an accumulation of round cells beneath the intima was observed, in another case cellular proliferation of the outer coat was demonstrated, and finally in still another case the walls of the capillaries in which the outer coat showed cellular proliferation, had undergone hyaline degeneration.

The vascular changes described by Hektoen, which were present in all of his 9 cases, consisting of a primary endarteritis and a phlebitis causing thrombosis and obliteration, were not uniformly present in my cases.

Hektoen believes that this primary endarteritis is due to the implantation of the tubercle bacilli upon the intima. He describes: (1) a diffuse form in which proliferation of the epithelioid cells occur between the elastic lamina and the endothelium; (2) isolated miliary tubercle upon the intima, either singly or in conjunction with diffuse endarteritis, and (3) diffuse changes which proceed from periarterial foci.

He quotes the works of Huguenin, Huttenbrener, Cornil and Hirschberg, who observed tuberculous endarteritis which was looked upon as primary and due to the implantation of the tubercle upon the intima.

Rindfleisch, Ziegler, Birsh-Hirschfeld, Lancereau, Baumgarten, and Guernieri and others believed the bacilli was localized in the adventitia, and Guernieri and Baumgarten described an endarteritis which they regarded as secondary. (Hektoen.)

Bloodvessel changes have been described by Raymond,[32] who found in the majority of the vessels of the pia an infiltration of the sheaths. Vascular changes have been also described by Diamond and Lartat-Jacob and Sabareanu. In Dreher's case proliferation of the intima went on to obliteration of the lumen, and there was also perivascular infiltration.

Ettinger said, on the contrary, that the absence of obliterating arteritis was striking and that the walls and especially the external and middle coat are a little thickened but uniformly so, and that the infiltration of the round cells is regular.

Encephalitis. A slight diffuse encephalitis is always present in these cases according to Oppenheim.

Distention of the perivascular spaces was noted in 7 of the cases under discussion. In 3 cases there was marked round-celled infiltration of the superficial layer of the cortex, and in another capillary hemorrhage was found. The cortex was rarefied in 2 cases and the cerebellum was the seat of a marked encephalitis in 1 case.

In this connection the work of Lhermitte is interesting. He described tuberculous meningo-encephalitis and believed it impossible to decide in a great many cases based upon the histological findings whether the condition was one of tuberculous encephalitis or a simple (banale) inflammatory reaction. He found in these cases that the plasma cell was present in the cortex.

Plasma Cell. Studies of the plasma cell in tuberculous meningitis have not been made to any extent until within the last ten years. Wolf[18] believed they were regularly present in tuberculous meningitis and believed further that degenerated plasma cells were

found in all cases though in varying amount, and raised the question whether these did not have some connection with regressive metamorphosis. Gehry[19] believed that the plasma cell played an important role in the regressive changes in the nerve elements.

The role of the plasma cell in vascular tuberculous meningitis was described at length by Diamond, who found them present in chronic and acute cases of tuberculous meningitis.

They were not found in all of the cases reported in this paper. In 6 cases, however, they were present to a greater or less extent.

As to the origin or significance of the plasma cell this paper does not strictly pertain. Sufficient to say that much has been written on the subject, and whether the plasma cell originates from the lymphocyte as Krompecher,[20] Marschalko,[21] Justi,[22] Joannovics,[23] Porcile,[24] Councilman,[25] Herbert,[26] Jadossohn,[27] and Schotlander[28] claim, or from the endothelial cell, as is believed by Unna,[29] Ehrlich,[30] and others, is still a matter of dispute.

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DISCUSSION

Dr. B. F. Stahl: I have nothing to offer in discussion except the unusual record of a case of tuberculous meningitis with recovery. In my service at the Philadelphia General Hospital I had under my care some time ago a man who unquestionably had tuberculosis, and who had all the symptoms of meningitis. From the statistics we, of course, expected a fatal termination. A very distinguished medical authority has said that these cases never recover, and that the cases so reported were not true tuberculous meningitis. The first essential in proving the existence of tuberculous meningitis is to examine the spinal fluid. In this case the bacilli were very much in evidence. The man after becoming convalescent from the meningeal symptoms was sent to White Haven, where the tuberculosis was treated and from there he was discharged as cured. He is still living and well two years after his discharge from White Haven. I wish to put this case on record, and at a later time will offer the case in full detail.

Dr. Rhein (closing): I have nothing further to say, except that the cases that I reported were tuberculous meningitis as we find it in children. I think the case reported by Dr. Stahl was in an adult. I did not understand whether it presented the marked symptoms seen in children. It probably did not, and this would rather tend to support my belief that tuberculous meningitis in children is a mixed infection, and that if we had only the tubercles themselves to deal with that the prognosis would be less grave than when complicated by infectious processes due to other microörganisms.

ILLUMINATING GAS POISONING[1]

By M. H. FUSSELL, M.D.

AND

ROBERT S. McCOMBS, M.D. (by invitation).

During the last ten years it has been the privilege of one of us to observe about one thousand cases of illuminating gas poisoning through his connection with the medical staff of the United Gas Improvement Company. The medical records of this company have also been tendered to us for purposes of study. Many of the cases observed have been repeatedly examined, some over a period as long as two years. The employees of the company in the largest generating plants, situated in Philadelphia, and members of the street gangs, who come more in contact with illuminating gas than others, have been under the direct observation of one of us throughout these years. During this time some of the men have been affected fifteen to twenty times; this has given the opportunity to study the so-called cases of chronic gas poisoning.

It is obviously impossible in the limits of this paper to note the full classification of symptoms and actual statistics of our studies. We will, however, attempt to give a complete summary of the results based upon our own observations and study of the records.

The toxic action of illuminating gas has been fully described by such authors as Sellman, Haldane, Edsall, Crile, and Lenhart. To summarize: The carbon monoxide present in illuminating gas is the poisonous principle, the other constituents of illuminating gas used to increase the candle power are non-toxic. Carbon monoxide has an affinity for hemoglobin three hundred times greater than oxygen; it excludes the oxygen; the resulting compound is not

[1] Read May 1, 1912.

perfectly stable so that when oxygen is present in very great excess the carbon monoxide-hemoglobin will be decomposed and oxy-hemoglobin formed. While oxygen cannot directly displace the carbon monoxide, it has been established that dissociation of the carbon monoxide-hemoglobin occurs under these circumstances, and that the carbon monoxide is expired as such, no noteworthy amount being oxidized into carbon dioxide. At death as much as 60 to 80 per cent. of the hemoglobin in the circulation will be in combination with carbon monoxide. The resultant symptoms are due to a diminution of the supply of oxygen to the tissues of the body. Other than this there does not seem to be any direct toxic action in the human being. There have been two instances in Philadelphia in which large numbers of plants have been destroyed from leaking gas in greenhouses. In our experience man is killed through asphyxiation alone. At first there is a stimulation of the central nervous system, followed by paralysis, the order being brain, spinal cord, and medulla.

For purposes of convenience the various symptoms of illuminating gas poisoning have been divided into three stages; this is our own classification, and while it is not claimed to be scientifically accurate, it was conceived with the idea of giving the employees definite divisions for the application of their first aid methods.

Symptoms. The first stage continues until the patient looses consciousness. During this period the following symptoms will develop: Cerebral excitement and dilatation of the cutaneous vessels, occasionally cyanosis will be noted if the poisoning is very acute, the venous blood being practically free from carbon monoxide; dizziness; headache; pains in the extremities; muscular twitchings; slow pulse from stimulation of the pneumogastric; rise in blood pressure from stimulation of the vasomotors; there is a feeling of constriction of the chest, rarely actual dyspnea; wide dilatation of the pupil; nausea and vomiting. Late in this stage there is a feeling of general weakness; faintness; languor and at times stupor. At times there is delirium. The temperature is normal or slightly elevated.

The second stage begins with syncope and ends with apnea.

The patient is unconscious but breathing, the respirations are rapid and stertorous with a corresponding increase in the pulse; the blood pressure falls; the temperature is elevated, at times reaching degrees of hyperexia; there may be loss of sphincter control and of some of the reflexes, although this is not constant. At times there are general tetaniform convulsions. Some cases show only a profound state of coma without the above mentioned symptoms; one case we saw in consultation had persisted for nine days; this man had not been properly treated according to our present knowledge. Toward the end of the second stage there is general muscular rigidity and a weak, rapid pulse, at times as high as two hundred; the jaws may be stiff and the tongue swollen; the peculiar cherry red markings, so characteristic after death, may make their appearance on the skin of the neck, trunk, and thighs. The spectroscope will show the presence of carbon monoxide in the blood. The respirations gradually become weaker until there is apnea.

In the third stage the patient is comatosed and respirations have ceased. A very rapid, weak, intermittent heart action is all that indicates the presence of life. All other symptoms of asphyxiation are present except cyanosis, which is usually absent or masked by the cherry red markings of the skin. The presence of carbon monoxide in the blood may be determined by the spectroscope. They may have the muscular rigidity mentioned before.

Death. If the skin surface is not discolored, within an hour or two it will be deeply marked especially in the dependent parts with the bluish or cherry red areas. Likewise, if not present at death, the toes will become fully extended and the hands assume the tetany position. This extension of the toes and fingers seems to appear earlier in children than in adults after death. The organs are deeply injected, especially the spleen; there are scattered small hemorrhages and at times cerebral changes (softening), symmetrical in many cases. The blood is pink in dilute solutions and shows the presence of carbon-monoxide by the spectroscope.

The test suggested by Hoppe-Seyler, namely, that blood containing carbon monoxide treated with double its volume of a solution of sodium hydrate yields a beautiful red color when spread

on a porcelain plate, while ordinary blood under the same conditions is changed into a dirty brownish mass, which exhibits a trace of green, has not given very satisfactory results.

A fairly accurate test is the action of the blood when it comes in contact with the atmosphere, if it immediately turns scarlet carbon monoxide is probably not present to any great extent, as its presence retards the formation of oxyhemoglobin. The spectroscope, however, is accurate.

The length of time necessary for a person to pass from one stage into the other is very uncertain; two factors determine this—first the amount of gas inhaled and second the personal idiosyncrasy. Under ordinary conditions a man can breathe a high degree of carbon monoxide for two minutes without symptoms; some of the men can stand it for five minutes. There are some exceptions to this rule; we have had men pass almost immediately from the first stage into the third stage after inhaling a small quantity of gas. One can never be sure that an individual who is apparently only slightly affected will not pass into the second or third stage very suddenly and without premonitory symptoms. This is not common, but a person suffering from the effects of gas should be constantly watched. Another phase is shown by the following: A young colored girl who had been exposed to a very mild degree of carbon monoxide, and apparently had no symptoms whatsoever, at the end of twenty-four hours developed stupor, severe pains in her extremities, and a temperature of 102°. In the meantime she had not been within several blocks of the leak. These cases are the exception not the rule. If a patient is breathing well when rescued the blood will be free of carbon monoxide in about three hours and the danger is over in about one hour. When respirations are shallow the time is lengthened.

Sequelæ. Symptoms that persist after the blood is free of carbon monoxide are referable principally to the nervous system. They include neuralgia, nervousness, insomnia, pain, intention tremors, loss of sexual power, loss of continuity of thought, delirium, confusional insanity, transient hemiplegia, and prolonged fever. There have been a few cases of bronchopneumonia. I have

not seen any cases of multiple sclerosis, ocular changes, nystagmus, paralysis of the recti, protrusion of the eyeball, nor gangrene. All of the sequelæ mentioned have eventually cleared up entirely within a period of six months; they constitute less than 2 per cent. of all the cases and are almost entirely confined to advanced second stage cases and third stage cases. Any method which tends to shorten the period during which the blood is impaired will tend to prevent the late symptoms due to tissue injury. There have been individuals poisoned who have been suffering at the time from chronic organic involvements of almost every description, many pregnant women are included in these statistics, also patients with tuberculosis and several who were in the midst of typhoid fever. None suffered any permanent bad effects and no miscarriages have occurred; the children when born have all been normal. Sequelæ are more likely to occur in those of advanced years.

The possibility of sequelæ developing sometime after the poisoning must be borne in mind, but all such cases in our experience have cleared up within six months.

Gilman Thompson considers it a bad sign if leukocytosis, which is usually present, be of a high degree.

Chronic Gas Poisoning. Examinations of the blood of employees who are constantly in contact with carbon monoxide, and of some who have been repeatedly poisoned have been made. The average count shows a polycythemia. This is in accord with the report of the investigating committee authorized by the Illinois Legislature and published in the *Journal of the American Medical Association*, also with the two cases reported by Reinhold.

We could not find any muscular weakness in the men examined; some of them had been employed in this line of work for twenty years and are now among the strongest and healthiest individuals we know. As a rule they pay no more attention to first-stage gas poisoning than they would to a headache. The estimation of muscular weakness by comparison with men in other occupations is difficult to obtain and is unreliable at the best.

We have seen no accumulative action of carbon monoxide unless the polycythemia may be an evidence of it. Reported symptoms

such as irregularity of the heart, slow pulse, anemia, lack of concentration, poor memory, cardiac dilatation, splenic enlargement and pleural effusions have not been observed.

Evidently the increase in the red blood corpuscles is to compensate for the presence of small quanitites of carbon monoxide so that the normal supply of oxygen to the tissues can be maintained.

Prophylaxis. It is not the purpose of this paper to belittle the poisonous nature of carbon monoxide; however, the fact remains that deaths result in nearly all cases from preventable causes.

It should be noted in comparing this statement with various reports of accidental deaths from carbon monoxide poisoning, which amounted to 624 in twenty-three years in the State of Massachusetts as reported by Sedgwick and Schneider, that the cases may be divided very sharply into two classes of accidental poisoning.

1. Those in which the accident consists in a direct break in a pipe in a room; failure to close the gas jet (the usual method); or by other accidental causes of a similar nature, all of which produce contamination of the atmosphere with a high degree of carbon monoxide in a short time.

2. Accidental cases due to street leaks; due to cracking of the mains by frost, the unequal contraction of frozen ground will at times break the largest and strongest pipes made. The escaping gas cannot filter through the superimposed earth and works its way through water and rat holes or along service pipes into the cellars. The odor of gas is always noticeable. In Philadelphia there has been only one instance where deaths have resulted from the second class of cases, and these were preventable.

Suicidal cases need not be considered, they make up approximately one-half of all fatalities.

The great majority of deaths were preventable if the individuals had slept with open windows, had paid attention to leaking gas and had reported it immediately to the company, and of course if they had properly closed the gas stop.

Treatment. The essential of treatment consists in the inhalation of oxygen, being under pressure where possible. The object

of all plans of treatment is to supply oxygen in sufficient quantities to displace the carbon monoxide in combination with the hemoglobin.

First Stage. In first-stage cases fresh air combined with mild stimulation, such as aromatic spirit of ammonia, repeated if necessary, should be administered. Oxygen inhalations are indicated in all stages. The nausea, vomiting, and headache are the most troublesome symptoms. The greater part of the gastric symptoms seem to be due to actual distention of the stomach with gas, no doubt swallowed in retching and with the gas-impregnated mucus from the nasopharynx. Some effervescing salt relieves these symptoms, the patients feeling much better after eructating or vomiting. It is due to this relief from nausea and gastric distention that a peculiar remedy for gas poisoning obtained its reputation among men working in gas as an antidote: it is an effervescing birch beer called "Weiss Beer." We have substituted effervescing phosphate of soda. There is no antidote for gas poisoning other than oxygen. The headache usually persists for twenty-four to forty-eight hours and may be relieved by any of the drugs used in this condition. Violent exertion is to be avoided; men who have become aggressively delirious have collapsed.

Second Stage. The patient is unconscious but breathing. It may be necessary if the respirations are not stertorous to assist the respiratory action. We instruct the men to apply the Howard method (compression of the lower part of the chest in rhythm with expiration). Oxygen must be administered preferably under pressure. Haldane placed animals (mice) in oxygen under a pressure of two atmospheres, so that simple solution of oxygen in the blood serum was obtained sufficient to support life independently of the hemoglobin. Enough carbon monoxide was present to completely saturate their hemoglobin. Under these conditions the animals remained normal as to symptoms, showing that the carbon monoxide had no direct toxic action. When the pressure was removed and the mice put out in the air they died with symptoms of asphyxia.

Various mechanical devices for administering artificial respira-

tion have been tried but until lately all were unsatisfactory. At present we are using the "pulmotor." This machine automatically adjusts itself to the individual capacity of the lungs, without danger of rupture, and maintains a mixture of oxygen and air (60 per cent.) under a constant pressure of five atmospheres. It maintains artificial respiration perfectly. We have reports from all over the country relative to its efficiency. Whenever possible it should be used.

Medicinally the patient should be freely given hypodermics of stimulants, such as camphor, caffeine, digitalis, and strychnine. Heat should be applied when indicated, and as there are several cases in which the persons have collapsed when taken out into cold air, it is always better to start treatment in a warm room. A very important adjunct to this plan of treatment is the massaging of the muscles, the increase in the hemoglobin in the general circulation accomplished by it often promptly restores the normal oxygen balance.

The above methods are usually followed by prompt recovery. If they are not successful, venesection with the introduction of normal salt solution should be employed. Personally two cases have come under our observation at the Philadelphia Polyclinic Hospital in which this method was followed by rapid and complete recovery, although there was some mental confusion in both cases for some time. Favorable reports of cases treated by this method are made by Jersey, Wilkie, Schruber, and von Gordon, eight in all, and including the two mentioned above make ten. Crile and Lenhart report five deaths at the Cook County Hospital, Chicago, in which this method was administered. We believe it should be employed when the patients are in fairly good condition when rescued, that is, a fair pulse, stertorous respirations, unconscious, and with no immediate signs of collapse. However, the "pulmotor" has made its necessity less frequent. Venesection and transfusion of defibrinated blood was practised by some clinicians in the seventies, those for and against this method were about equally divided. The best method when the "pulmotor" is failing is the direct transfusion of blood as practised by Crile and his followers.

His experiments reported in the *American Journal of the Medical Sciences* led him to conclude that blood transfusion seems to be of greater therapeutic worth than the other measures considered. It has been used in two cases in this city, both of which made a prompt recovery after other methods had failed. The introduction of fresh hemoglobin which will combine with oxygen and which will not be contaminated by the carbon monoxide already held in a combination with the patient's hemoglobin is a sure way of restoring the normal oxygen balance. Transfusion should be done immediately if the patient is in the advanced second or in the third stage and fails to respond to the ordinary treatment.

THIRD STAGE. The patient is not breathing and is unconscious. Artificial respiration, oxygen, stimulation, and heat are imperative. Transfusion is indicated. Our employees use the Schafer or "prone pressure" method of artificial respiration. The "pulmotor" has maintained artificial respiration in this stage.

We have had excellent results from our instruction to employees, artificial respiration having been maintained for six hours with subsequent recovery of the patient. In one instance two physicians had pronounced the man dead, but the employees persisted and the patient finally recovered.

PROGNOSIS. If the above methods of treatment are carried out and the patient is not dead when discovered practically all cases should recover in forty-eight to seventy-two hours from the immediate effects of the gas, no matter how concentrated the carbon monoxide causing the poisoning. The great difficulty in treatment has been the failure of both laity and physicians to realize that inhalation of oyxgen under pressure combined with blood transfusion in severe poisoning will save practically all the cases.

Of 39 cases reported by Edsall occurring at the Episcopal Hospital in Philadelphia, mostly suicidal in intention, 34 fully recovered and most of the fatal cases were due to sequelæ.

DISCUSSION

Dr. George M. Dorrance: Transfusion as an adjunct in the treatment of cases of gas poisoning is not new. It was used years ago at the Pennsylvania Hospital. With the use of the pulmotor which Dr. McCombs has shown, I think transfusion will seldom be required. If transfusion is required the vein-to-vein method is the simplest and the technique the easiest. The reason transfusion is not more frequently employed is because the average surgeon fails in about 25 per cent. of the cases attempted.

Dr. John B. Roberts: Will Dr. McCombs be kind enough to tell us as a matter of interest whether there is any general use of this same form of illuminating gas throughout the country. I understand that the gas used here now is much more dangerous than formerly because of the recent combination of coal gas with the so-called water gas. We as laymen in gas matters, would be glad to know why the term "illuminating gas" is used in his paper. In other words, does he mean by "illuminating gas" only the unknown mixture of products used for illumination in Philadelphia.

Dr. G. E. de Schweinitz: The Chair has examined a few patients suffering from illuminating gas poisoning without finding any noteworthy ocular symptoms. They have been described, however, in carbonic oxide poisoning, and a very interesting phenomenon is the development of hemianopsia, probably depending upon a lesion of the occipital lobes. It seems strange that retinal hemorrhages are not more frequently seen, perhaps because they have not been searched for with sufficient frequency. It is probable that careful examination would develop the presence of alterations in the visual field in illuminating gas poisoning, because in carbonic oxide poisoning concentric contraction of the visual field and loss of the color sense have been observed.

Dr. William R. Nicholson: I should like to ask Dr. McCombs whether such an instrument as he has exhibited is prepared for use in the newborn with threatened asphyxiation?

Dr. McCombs, closing: In answer to Dr. Nicholson, I have seen here in Philadelphia one pulmotor of the size to be used for a baby. It was not automatic, and I should say was about half the size of the one exhibited here. The pressure of a column of water eight inches high is enough to expand a man's lungs.

In regard to Dr. Roberts' question, so far as I know, the illuminating gas is more or less of a trade secret. I should say that 75 to 80 per cent. of all gas plants have a combination of both water and coal gas reënforced by water vapor and heavy oil for candle-power purposes. Of course the gas is put through a process to take out the sulphates and ammonia, and the standard is about the same in most of the large cities.

THE SPINAL CORD IN PERNICIOUS ANEMIA, WITH THE REPORT OF AN INTERESTING CASE OF FAMILY INVOLVEMENT[1]

By ROBERT N. WILLSON, M.D.

The clinical picture of pernicious anemia and the microscopic findings in the blood are so thoroughly understood at the present time that they may be passed over without comment even in a discussion of the changes found in the spinal cord in this symptom-complex rather than disease. It is well, however, to remember that there do exist spinal cord and perhaps brain changes in every case of true pernicious anemia, and that many cases that fail of prompt recognition owing to nervous symptoms, would at once become clear if this fact were kept in mind. The likelihood that both the clinical picture and the cord degeneration are secondary to and the direct result of a definite toxemia has also occurred to and been hinted at by many investigators. Yet, up to today, the nature and source of this toxin and its method of action are still matters of speculation, and even its avenue of approach and of attack are still to be determined. Lictheim in 1887, Putnam and again Dana in 1891, Dana in 1899, and later Pickett, Burr, Spiller, Camac, and in the last months Camp, have all described the combined sclerosis and degeneration of the cord which have come to be well-recognized features in not a few instances of pernicious anemia. Spiller has shown that at least occasionally the degenerative process extends into the brain axis. Pickett, and much later Marcus (1903), and in the current year Camp, have described cases of pernicious anemia in which there

[1] Read October 2, 1912.

became prominent the mental picture and the signs of paresis. There has not yet been reported, however, a case which has suggested so definitely as does that which forms the basis of this brief study the likelihood of a direct causal relation between the intestinal tract and both the peculiar anemic state and the degenerative process in the spinal cord. The spinal cord lesions of pernicious anemia, as already repeatedly demonstrated, consist of a combined sclerosis of the posterior and lateral columns. Occa-

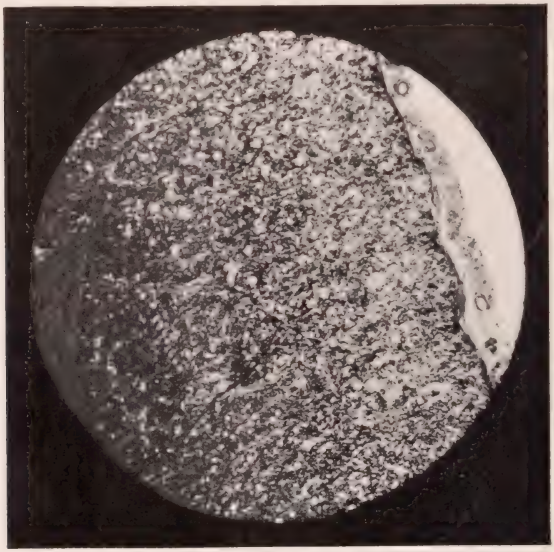

FIG. 1.—Lumbar cord, white matter, posterior columns, showing spaces from which nerve fibers have dropped out.

sionally the posterior columns are alone affected, and there attends a definite clinical picture of tabes dorsalis. The lateral columns never constitute the only field of involvement. There is little tendency to implication of the gray matter or of the nerve roots, or to contraction and shrinking of the cord such as are seen in true tabes. Hemorrhages are frequently in evidence and also processes that can perhaps best be explained upon the basis of thrombosis of the spinal vessels. Usually the degeneration appears to start in the upper dorsal or in the lower cervical cord, the posterior

columns sustaining the earliest and most severe attack. In the writer's case the lumbar cord showed the most marked evidence of disease (Fig. 1). The lateral columns in most cases are also sooner or later involved, and later in the course of the intoxication the anterior horns show both clinical and microscopic signs of disease. There is more or less proliferation of the neuroglia, with rarefaction of the nerve fibers, and often a complete dropping out of the latter. The cells in the gray matter often show degenerative changes, and may be pigmented.

FIG. 2.—Lumbar cord, cells in anterior horns. Some cells are swollen, some have lost their processes, others show vacuolization.

Camac and others have described cavity formation in the cord probably due to edematous softening and consequent degeneration. In Camac's case the cavity corresponded accurately to the distribution of a branch of the anterior spinal artery, and apparently a thrombosis in one of the small veins was the original cause of the excavation. In a word, it may be said that the pathological process consists of a degenerative vacuolization of the nerve cells, accompanied or rather succeeded by a replacement gliosis.

A considerable number of cases have been reported in which the anterior horns were involved to such an extent that the clinical amyotrophic result was confused with emaciation and obscured the diagnosis. This is an occurrence of the late stages of the intoxication, however, and in view of our present information it would seem as though this confusion should no longer be necessary, even though the blood picture may not yet have assumed the definite character of pernicious anemia and of pseudocombined sclerosis (Fig. 2).

The case which forms the subject of this paper is of interest from more than one direction.

The patient, A. C. W., was a single woman, aged forty-three years. Her family history was very striking, and was, in brief, as follows: Her father died at the age of sixty years. He had suffered from "asthma," dropsy, and "had a stroke." The picture is at least suggestive of a uremia. He had also had hemorrhages from the bowel, though not recently before his death. The patient's mother is still living, aged seventy-nine years,and has aortic insufficiency with cirrhosis of the liver. One of the patient's aunts on the mother's side had hepatic trouble, and suffered from angina pectoris and Bright's disease. Another aunt on the same side died of pernicious anemia. On January 17, 1907, her blood record was as follows: Red corpuscles, 955,000; leukocytes, 3000 per cubic millimeter; hemoglobin, 32 per cent.; color index, 1.77. A number of megaloblasts and a few normoblasts were noted. A brother of the mother died, aged sixty-eight years, of mental trouble. One sister died a year ago of tabes in a grave anemia (seen by me). Two of the patient's brothers are living, but are invalids. One of these shows a marked atrophy of the muscles of the right side consequent upon early anterior poliomyelitis, also some renal and bladder involvement. This brother has had glaucoma of one eye, and was operated upon for this condition ten years ago. Another brother has kidney disease with "puffing under the eyes."

Previous History. The earliest record obtainable of the patient indicated normal health up to the sixteenth year, after which she showed a marked tendency to severe colds. She had two attacks of congestion of the kidneys between the nineteenth and twenty-ninth years, and suffered a pleurisy also during this period. *The severe colds in the head and throat have continued up to the present time, and have always been accompanied by indigestion, which has gradually developed into the severe mucous enteritis that preceded her death.* Her menstruation was always irregular in time and quantity.

In 1911 the patient was taken to the West Philadelphia Hospital for Women for an intestinal attack with eructations of gas, and with considerable abdominal tenderness. In October, 1911, she experienced severe pain under the right breast increased by pressure over the gall-bladder. This attack began with eructations of gas, lasting for hours.

She was admitted to the Women's Hospital at this time, and through the courtesy of the institution and of Dr. Williams I am able to present the notes of her illness at that time. In January, 1911, she had menstruated very profusely; then again in March, then in June, July, and August, but not after that time. In February she had considerable bleeding from the bowel, with intense soreness over the abdomen. She also suffered much from cardiac arrhythmia and palpitation, both evident to herself. While in the hospital she had much nausea and gastric distress, which were relieved at times for a day or two, but returned in sudden acute attacks. During these attacks the face was flushed, and the pupils unequal. Most of the time the pupils were markedly contracted. Following the excitement of such an attack the temperature rose, but was usually normal. Pelvic examination was negative. Abdominal examination showed ptosis of the stomach and intestines, with areas of tenderness on palpation about the umbilicus and over the gall-bladder. The heart and lungs were normal.

It is interesting to note that at this time the red corpuscles numbered 6,500,000, and the leukocytes 15,900. The feces showed much mucus, but no occult blood.

An examination of her eyes by Dr. Stratton in 1901 showed: "(O. D.) media clear, bright reflex from vessels, deep central physiological cup, and the edges of the nerve hazy; (O. S.) media clear, nerve head hyperemic, otherwise same as O. D." "In 1909 muscular insufficiency, both vertical and lateral, refraction findings and vision practically the same as in 1901."

Current History. The patient was first seen by me in consultation with Dr. R. R. Williams on December 21, 1911. She had for a year been suffering from gradually increasing disability and discomfort in the form of weakness and lightning pains in the extremities, in addition to her gastro-intestinal trouble of long standing. She had also been experiencing with increasing frequency severe gastric crises, comprising pain, nausea, and vomiting. A constricting girdle pain was described by the patient herself as severe about the level of the ensiform cartilage. There was a frequent and copious eructation of gas and considerable blood was passed at times from the bowel. During the last weeks an increasing and finally a very marked pigmentation of the skin was noticed until the entire body was dark brown, while over the abdomen, especially around the umbilicus, there was an area almost black in color. Over the

buttocks and the posterior surfaces of the upper arms and of the elbows there was a similar intense pigmentation. There was no emaciation. The patient could stand but would fall at once when the eyes were closed. She complained bitterly of intense tingling and pain in the extremities, especially in the hands and fingers, and described an aura of this type preceding the gastric crises. During the last ten days of her life large hemorrhagic blotches appeared on the elbows and over the knuckles of both hands. These rapidly changed to hemorrhagic blebs, which soon ruptured, leaving extensive ulcerations in their place.

On *physical examination* the skin surface was as already described. The mucous membranes were blanched and extremely anemic, and here and there over the buccal mucous membrane were patches of brown pigmentation. There was no jaundice. Pressure over the sternum and long bones elicited marked tenderness. Both pupils were very small and completely fixed. There was no reaction to light, and a very slight response to distance accommodation. Both patellar reflexes were completely absent, as were also the Achilles tendon and the plantar reflexes. Babinski's reflex was not present on either side. There was a complete loss of station with the eyes closed, the patient falling unless supported.

The finer acts of accommodation and coördination were all embarrassed and imperfect. While being examined the patient had several attacks of severe pain, mainly in the lower limbs, undoubtedly lightning pains. There were areas of moderate hyperesthesia, as well as less extensive areas of complete anesthesia scattered over the body. The heart and lungs appeared normal, except for indicating a general debility. The abdominal organs, including the liver, spleen, appendix, uterus, and adnexa, showed no abnormality on palpation. The abdomen was generally sensitive, but presented no localized area of involvement. The *urine* contained a small quantity of albumin and a few hyaline and hyalogranular casts. The *feces* contained much blood, mucus, and undigested food, but no parasites or ova. The *blood* examination gave hemoglobin, 50 per cent.; red blood corpuscles, 3,060,000; leukocytes, 8000. There was considerable poikilocytosis, polychromatophilia, and there were found occasional nucleated red cells, mainly normoblasts.

Autopsy. The body was examined on January 8, 1912, the day following the death of the patient. The skin and mucous membranes were found pigmented as already described, also the large sloughing areas over the elbows and knuckles which had taken the place of the hemorrhagic bullæ. On opening the thorax the only organ that presented any special abnormality was the heart, which was small, very flabby, with an evident insufficiency of all its valves. Just outside the aortic ring, also around the opening of the right coronary artery and over the surface of the left auricle, were patches of atheromatous deposit. In the wall of the left

auricle and in the posterior cusp of the mitral valve were two atheromatous and calcified nodules. The abdominal organs were all intensely anemic and showed the changes characteristic of pernicious anemia. In the kidneys were evident many distended and injected vessels. The adrenal glands appeared merely anemic. The intestines showed an extensive inflammatory process with considerable erosion of the duodenum. The colon was eroded from the cecum to the anus, and in places there were found areas of shallow ulceration. The stomach was dilated, its mucous membrane considerably injected, its muscular wall very thin, but there was no ulceration present. The brain showed no abnormality. The dorsal, lumbar, and sacral portions of the spinal cord were removed.

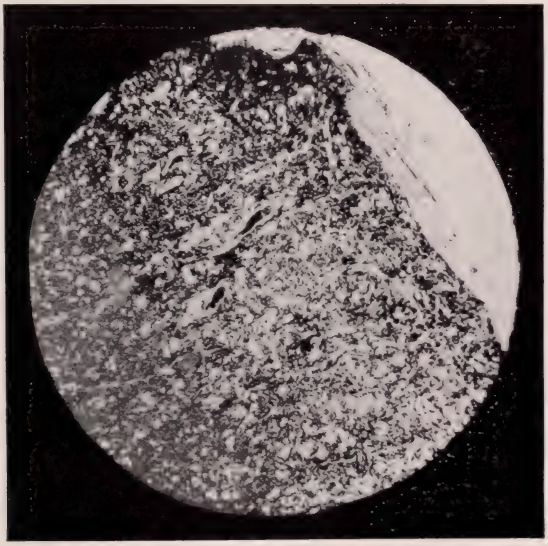

FIG. 3.—Upper thoracic cord, white matter, posterior columns. The same changes are present as in the lumbar cord.

Dr. William G. Spiller, who very kindly examined the sections for and with me, coincides in the following report upon the spinal-cord involvement. Sections were examined from various portions below the cervical region, including the dorsal, lumbar, and sacral segments. Degenerative changes were found throughout the white columns. These resulted in a dropping out of nerve fibers, leaving numerous holes in place of the fibers which had disappeared. This resulted in the characteristic lacework appearance which is so beautifully shown in the slides (Fig. 3). There was very little proliferation of neurogliar tissue. There was

approximately a similar and equal involvement of the posterior and lateral columns. The nerve cells of the anterior horns of the lumbar region were considerably affected. Many were swollen and in many pronounced chromatolysis had occurred. The degenerative changes were those which are most usually seen in the tissues of the spinal cord in pernicious anemia.

The foregoing history and autopsy protocol present special interest from the standpoint of the possibility of establishing a definite etiology for the clinical symptoms and for the pathological lesions as found during life and later under the microscope. Unfortunately the patient was not suspected of developing either pernicious anemia or pseudocombined sclerosis at a stage of the disease at which there may have been present spastic symptoms (at least exaggerated reflexes), and at which she might have suggested the presence of involvement of the lateral columns. The spinal picture from the time it came under the notice of the writer was definitely one of posterior column involvement plus, during the last days of life, involvement of the anterior horns with suggestive trophic changes.

Very striking, however, is the clinical indication of a pathological intestinal process dating back to childhood and terminating in the gastric crises which indicated perhaps the final influence of the auto-intoxication upon the spinal cord. The writer in a previous clinical communication called attention to the symptoms of intestinal decomposition and auto-intoxication comprising just a series of "colds" as the patient under consideration presented during her entire lifetime. In view of her entire freedom from involvement of the respiratory apparatus, as evidenced both by the physical examination and by the autopsy findings, there would seem to be little room for doubt that this individual for many years had suffered from a toxemia, metabolic in origin, and that the gastro-intestinal tract had been its manufactory. It seems evident that this toxemia led up to, even if not finally demonstrated as being the direct cause of the pernicious anemia, of the spinal-cord degeneration and sclerosis, and of the fatal termination of the case.

Of no slight interest, moreover, is the series of instances in the patient's near and immediate family connection of pathological clinical pictures which suggested not only a definite relation between the pernicious anemia and combined sclerosis of this case and the father's arteriosclerosis and uremic "stroke," the mother's aortic insufficiency and hepatic cirrhosis, with the renal sclerosis and angina pectoris of one aunt, and the pernicious anemia of another, to say nothing of the death of a third aunt (all

FIG. 4.—Upper thoracic cord, cells in anterior horns. The same changes are present as in the lumbar cord.

three sisters of the mother) in an advanced stage of locomotor ataxia. A brother of the mother died of "mental trouble," the exact nature of which could not be determined. It does not seem at all unfair to consider the possibility if not the probability, of an hereditary luetic influence running throughout this long series of circulatory and nervous involvements (Fig. 4). Especially in that type of case in which the picture of locomotor ataxia has been prominent, or in which the mental phase of paresis has been

definite and prominent, there have been many recent attempts to demonstrate the syphilitic origin of pernicious anemia. As in the case reported by Camp, however, both the negative Wassermann test, the negative findings in the cerebrospinal fluid, and the absence of the Argyll-Robertson pupil, in many instances argue against a positive statement in this direction. C. W. Field has recently informed the writer of 4 cases of pernicious anemia studied by him of which one furnished a positive Wassermann reaction and 3 reacted negatively. He also writes of "7 cases of grave secondary anemia which from their blood picture resembled pernicious anemia very closely. Two of these gave positive reactions and both of these admitted having contracted the disease some years before. The 5 negative cases denied a syphilitic history and showed no signs of syphilis." A history such as that presented today, together with microscopic findings of the character noted in the sections of the cord now submitted, cannot fail to indicate the strong possibility, and perhaps the probability of the intestinal origin of not only pernicious anemia, but of the pseudocombined spinal sclerosis. Berger and Tsuchiga have apparently shown that in pernicious anemia there is in the intestinal mucosa a lipoid ten times more powerful as a hemolysin than the lipoid from the normal mucosa. This has not been shown, however, to have any influence upon the spinal cord. The relation of true tabes dorsalis to syphilis is too well understood to require any comment here. The working of syphilis through the gastro-intestinal tract, and its influence upon the blood and upon the spinal cord through a gastro-intestinal autotoxemia has not been so thoroughly considered, and is offered as a theory not without a certain measure of plausibility in the case under study. It is satisfactory at least, in meeting the requirements of this individual case, in which from childhood there were present signs of gastro-intestinal disability. The deep pigmentation is interesting from one standpoint only, that of its severe degree. Pigmentation is so common in pernicious anemia that ordinarily it calls for no comment, especially in cases that have been treated with arsenic. The hemorrhagic bullæ on the knuckles and elbows,

and the subsequent open ulcers, were unusual, and apparently combined the customary tendency to hemorrhage with distinct trophic change. It would seem as though the case was almost certainly one of pernicious anemia in and perhaps due to hereditary lues, and that the somewhat similar cases in two of the patient's maternal aunts were in close relation and to be attributed to the same etiology.

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THE TOPOGRAPHY OF THE CARDIAC VALVES AS REVEALED BY THE X-RAYS[1]

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For many years attempts have been made to determine definitely the exact spots on the anterior chest wall under which lie the valves of the heart. The method by which studies in this direction have been made has consisted of the thrusting of long pins into the thorax and then opening the latter and noting the points at which the pins pierced the heart. From data thus obtained diagrams were constructed which were supposed to depict the valves in their relation to the anterior chest wall. That this method is inaccurate is demonstrated rather forcibly by the lack of unanimity in the descriptions given by various authors. In the following excerpts we present the opinions of a number of authorities, and it is interesting to note that the more superficially located is the valve the more nearly do the different authors agree, while the deeper lies the valve the more widely do the writers disagree.

Aortic: Behind the junction of the third left costal cartilage with the sternum, one-fourth covered by pulmonary valves. *Pul-*

[1] Read October 2, 1912

monary: Behind the upper border of the third left costal cartilage and the adjacent part of the second left intercostal space. *Tricuspid:* Behind the left half of the sternum at the fifth costal cartilage and the fourth intercostal space. *Mitral:* Behind the

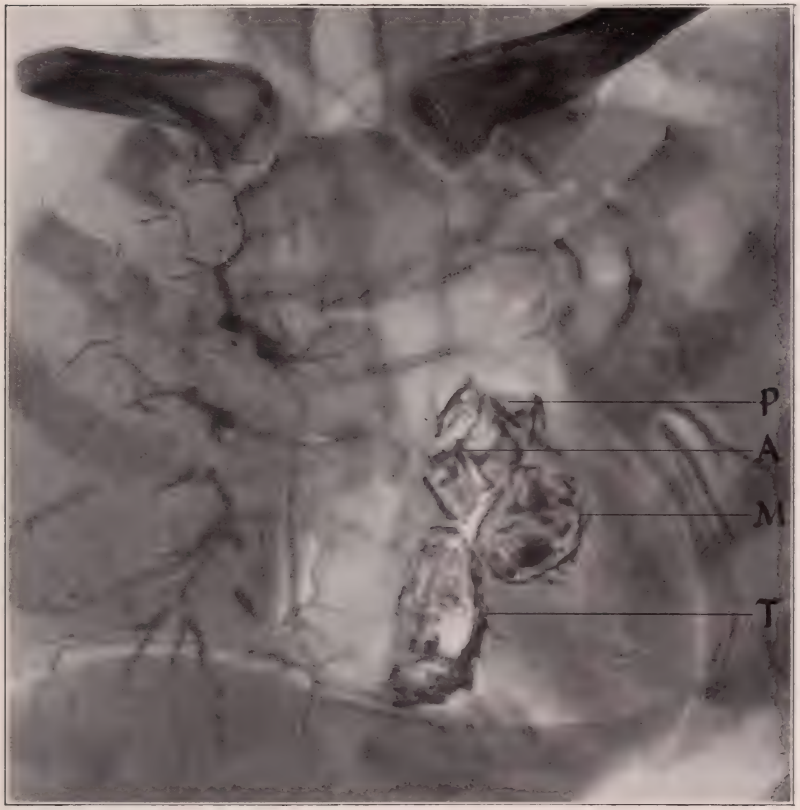

FIG. 1.—In this *x*-ray photograph every valve is shown with great clearness. On account of cardiac hypertrophy and sinistro-displacement, all the valves lie well to the left of the sternum. *P*, pulmonary valve; *A*, aortic valve; *M*, mitral valve; *T*, tricuspid valve.

left half of the sternum at the third intercostal space and at the fourth intercostal space.[1]

Aortic: Behind the sternal end of the third left intercostal space. *Pulmonary:* A little above and behind the third left

[1] G. A. Gibson, Diseases of the Heart and Aorta, 1898, p. 38.

chondrosternal junction. *Tricuspid:* Behind the middle of the sternum at the fourth intercostal space. *Mitral:* Behind the fourth costal cartilage at the left border of the sternum.[1]

Aortic: Slightly below and internal to the pulmonary on the level with the third space between the midsternal and the left sternal line. *Pulmonary:* Behind the sternal end of the third

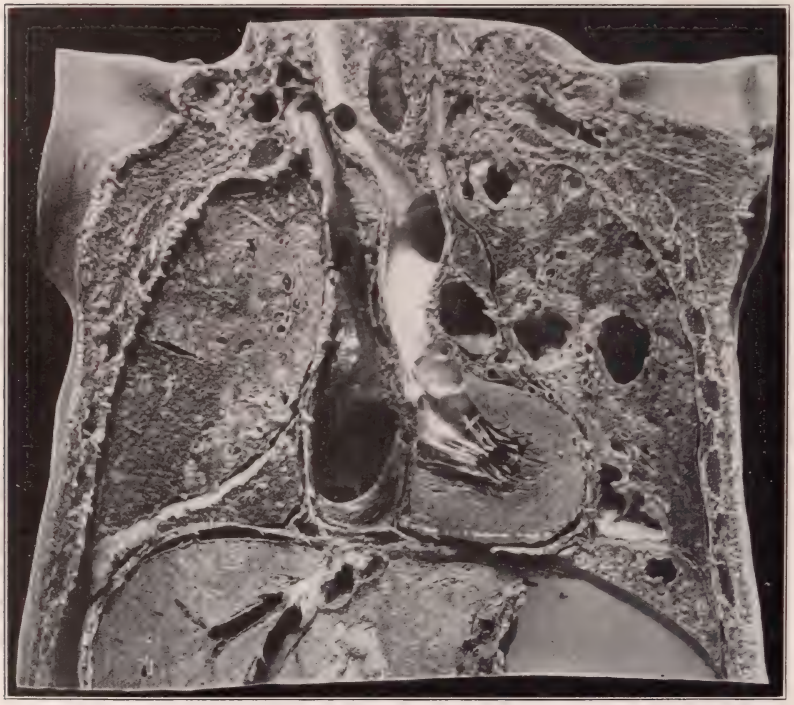

FIG. 2.—Frontal section of the thorax, the *x*-ray picture of which is shown in Fig. 3. The tuberculous cavities of the left lung are well shown in the *x*-ray plate.

left costal cartilage. *Tricuspid:* On the horizontal line between the two sternal ends of the fifth cartilage at the point intersected by a line drawn from the sternal end of the third left cartilage to the upper border of the sternal end of the seventh on the right. *Mitral:* Behind the sternal end of the third left space.[2]

[1] John B. Deaver, Surgical Anatomy, 1903, p. 409.
[2] de Nancrède, Essentials of Anatomy, 1911, p. 183.

Aortic: Lower border of the third left costosternal junction. *Pulmonary:* Upper border of the third left costosternal junction. *Tricuspid:* Just to the left of the midsternal line level with the fourth space. *Mitral:* Fourth left costosternal junction.[1]

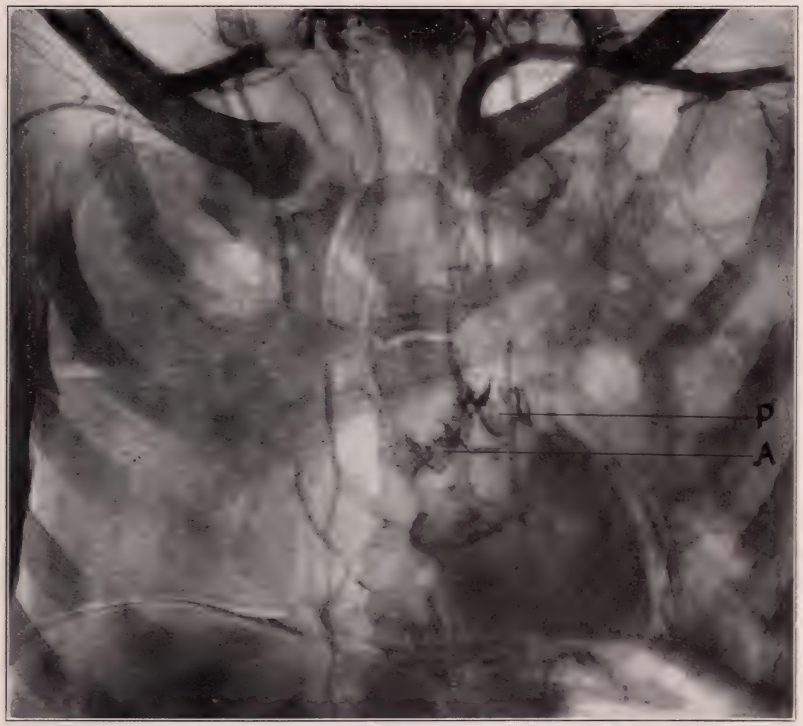

FIG. 3.—*X*-ray photograph of thorax, a frontal section of which is shown in Fig. 7. Of the cardiac valves only the semilunars show with clearness. The tuberculous cavities of the left lung and the internal mammary vessels are well demonstrated. *P*, pulmonary valve; *A*, aortic valve.

Aortic: The third left intercostal space. *Pulmonary:* Third left costosternal junction. *Tricuspid:* Midsternum at the level of the fifth costosternal junction. *Mitral:* Fourth left costosternal junction.[2]

[1] Henry Gray, Anatomy, Philadelphia, 1908, p. 559.
[2] C. Toldt, Atlas of Human Anatomy, New York, 1904, Part 5, p. 586.

Aortic: Lower border of the third left costosternal junction. *Pulmonary:* Upper border of the third left costal cartilage close to the sternum. *Tricuspid:* Behind the sternum at the level of the fourth interspace and anterior extremity of the fifth costal cartilage

FIG. 4.—Frontal section of the thorax, the *x*-ray picture of which is shown in Fig. 5. There should be noted the cut surfaces of the aorta, the pulmonary artery, and the left auricular appendix, all of which appear clearly in Fig. 5.

extending down to the right almost as far as the sixth chondrosternal junction. *Mitral:* Behind the left half sternum at the level of the fourth chondrosternal junction.[1]

[1] L. B. Rawlings, Landmarks and Surface Markings of the Human Body, London, 1908, p. 37.

Aortic: Opposite the inner lower border of the third left costal cartilage. *Pulmonary:* Upper border of the third left costal cartilage at its sternal junction. *Tricuspid:* Behind the sternum from the opposite fourth left interspace to the fifth right inter-

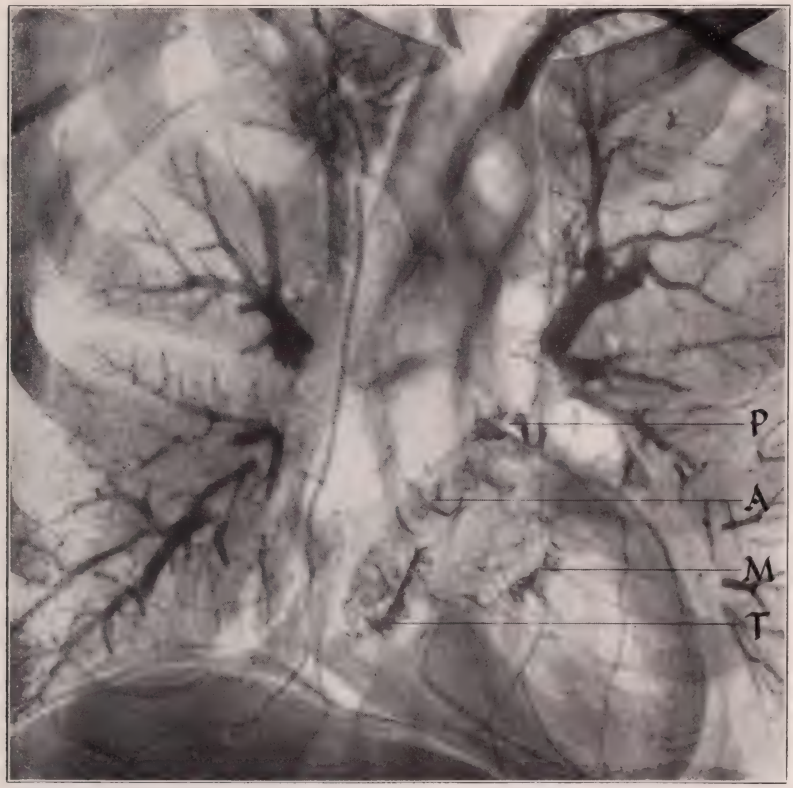

FIG. 5.—*X*-ray picture of the thorax, a frontal section of which is shown in Fig. 4. The valves in this plate are photographed rather faintly. They are more widely separated than is usually found, the condition being probably due to cardiac dilatation. The aorta, the pulmonary artery, the left auricular appendix (see Fig. 4), the pulmonary veins, and the internal mammary and coronary arteries are shown with great clearness. *P*, pulmonary valve; *A*, aortic valve; *M*, mitral valve; *T*, tricuspid valve.

space. *Mitral:* Left edge of the sternum at the level of the fourth left costal cartilage.[1]

Aortic: Behind the third intercostal space, close to the left side of the sternum. *Pulmonary:* In front of the aortic valve,

[1] C. R. Whittaker, Essentials of Surface Anatomy, London, 1912, p. 29.

behind the third left chondrosternal junction. *Tricuspid:* Behind the middle of the sternum, about the level of the fourth costal cartilage. *Mitral:* Behind the third intercostal space, about one inch to the left of the sternum.[1]

FIG. 6.—Frontal section of the thorax, the *x*-ray picture of which is shown in Fig. 7.

Aortic: Middle of the sternum, at the level of the third costal cartilage. *Pulmonary:* Second intercostal space, somewhat to the left of the edge of the sternum. *Tricuspid:* Midway between

[1] John H. Musser, Medical Diagnosis, 1900, p. 595.

the third left and the fifth right chondrosternal articulation. *Mitral:* Beneath the third left chondrosternal articulation.[1]

Aortic: Behind the left half of the sternum at the lower border of the third costal cartilage. *Pulmonary:* At the upper

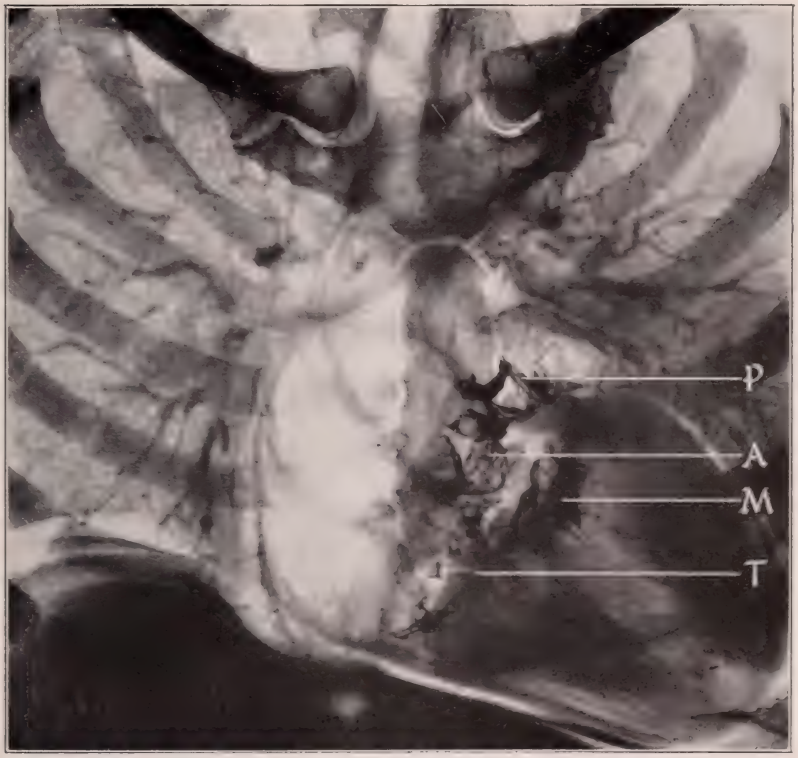

FIG. 7.—*X*-ray picture of the thorax, a frontal section of which is shown in Fig. 6. The individual leaflets of the pulmonary and aortic valves are shown with unusual distinctness. All the valves lie approximately one rib below what is described as their normal position. *P*, pulmonary valve; *A*, aortic valve; *M*, mitral valve; *T*, tricuspid valve.

border of the third left chondrosternal articulation. *Tricuspid:* From the midsternum at the level of the fourth costal cartilage to the fifth chondrosternal junction. *Mitral:* Behind the left half of the sternum at the level of the fourth costal cartilage.[2]

[1] Herman Sahli, Diagnostic Methods, 1905 (quotes Luschka), p. 247.
[2] J. C. Da Costa, Jr., 1911, p. 298.

Aortic: Behind the midsternum at the level of the third intercostal space. *Pulmonary:* Behind the third left chondrosternal articulation. *Tricuspid:* Behind the lower portion of the sternum at the level of the fourth intercostal space. *Mitral:* Behind the fourth chondrosternal articulation.[1]

Aortic: Behind the left half of the sternum, opposite the lower part of the third chondrosternal articulation. *Pulmonary:* Behind the third left chondrosternal articulation. *Tricuspid:* Behind the midsternum, opposite the fourth chondrosternal articulation and the fourth intercostal space. *Mitral:* Behind the left half of the sternum opposite the third interspace and the fourth chondrosternal articulation.[2]

Aortic: Behind the left half of the sternum a little below and to the right of the pulmonary. *Pulmonary:* Behind the sternal end of the third left costal cartilage. *Tricuspid:* Almost behind the midsternum, opposite the fourth interspace and the fifth chondrosternal articulation. *Mitral:* Opposite the sternal end of the third left interspace.[3]

Aortic: The third interspace close to the sternum. *Pulmonary:* At the junction of the third rib with the left side of the sternum. *Tricuspid:* Behind the sternum, near the middle line about the level of the fourth costal cartilage. *Mitral:* Behind the third intercostal space about one inch to the left of the sternum.[4]

Aortic: Behind the inner edge of the third costal cartilage, intercostal space, and contiguous portion of the sternum on the left side. *Pulmonary:* A little higher and more to the left. *Tricuspid:* Below and a little to the left, a line drawn from the inner end of the third costal cartilage on the left to the inner end of the sixth right costal cartilage. *Mitral:* Behind the third intercostal space about one inch to the left of the sternum.[5]

With the view of settling definitely these moot points the present study was undertaken. In the planning of our investigations the

[1] Joh. Sobotta, Atlas and Text-book of Human Anatomy, American Translation, 1906, ii, 172.

[2] D. J. Cunningham, Text-book of Anatomy, 1906, p. 789.

[3] G. A. Piersol, Human Anatomy, 1907, p. 692.

[4] H. Morris, Human Anatomy, Philadelphia, 1895, p. 972.

[5] J. Leidy.

problem naturally divided itself into two parts, (1) the preparation of the material so that conditions existing during life would be present for postmortem study, and (2) the depiction of such conditions with all possible sources of error eliminated. In order to secure these desiderata the following plan was evolved: Each cadaver on being delivered at the University of Pennsylvania was injected with 10 per cent. formaldehyde solution and then put into cold storage. When thorough freezing had been secured the head and neck, the upper extremities, and the lower part of the trunk were cut off. The thorax was then cut into frontal sections about one inch thick, the cuts being made parallel with the anterior surface of the chest. Each section was then cleaned up by removing all blood clots and saw detritus from the heart cavities and large bloodvessels. In this manner we avoided distortion, since all parts were hardened in the position which they occupied at death.

To depict the conditions present we adopted the following procedure: Each valve leaflet was carefully dried and then painted with the thickest possible lead paint. This was readily done, for the sections were so thin that all the valves were accessible, and in most instances it was possible to paint both surfaces of the entire eleven leaflets.

Finally, after the valves were painted, the sections were superimposed, with the anterior one below, and an *x*-ray photograph taken. In order to show with greater clearness the relations of the valves to the sternum and ribs, that section of the thorax which comprised the posterior portion and included the vertebral column, the posterior section of the ribs and the thick spinal muscles, was not used. By discarding this portion nothing was lost and much was gained in clarity, since the vertebræ and the posterior part of the ribs were eliminated from the pictures.

In the course of our investigations fifteen bodies were sectioned. It was soon evident, however, that a much larger number would have to be studied before any definite conclusions regarding normal relations could be reached, since many of the cadavers represented pathological alterations in the topography of the thoracic viscera.

Often the heart was displaced as the result of extensive pulmonary tuberculosis, or hypertrophy of the heart. Other lesions, such as pericardial effusions, atalectatic lungs, etc., furnished additional sources of error.

We therefore offer this communication in the nature of a preliminary report, and in demonstration of an entirely feasible and hitherto unemployed method of studying post mortem the topography of the heart valves in health and disease.

In conclusion, we wish to express our indebtedness to Dr. Henry K. Pancoast for his kindness in furnishing us with the *x*-ray photographs.

DISCUSSION.

Dr. Robert N. Willson.—It seems to me that this paper should not be allowed to pass without at least a word of congratulation from those of us who are interested and working in the same field. The study, on its face, has involved a tremendous amount of work. While no definite clinical results have been obtained, the paper points the way to the obtaining of information that has long been wanted. The mere fact, however, that only pathological chests were examined, there being but one normal chest in the series, emphasizes the point that pathological organs do not show even normal positions of their integral parts. The anatomical and frozen section work is beautiful and the demonstration of the abnormal location of the valves in dilated hearts, although not new, is quite clear. I have in my possession a number of children's hearts which are much larger than the average adult organ. In each and every instance the valves are far from their normal position. Only from the study of a series of normal hearts in their relations to normal lungs will it ever be possible to determine the normal positions of the cardiac valves.

TIC AND SPASM OF THE FACE; DIFFERENTIAL DIAGNOSIS; EFFECT OF ALCOHOLIC INJECTIONS[1]

By ALFRED GORDON, M.D.

Among various morbid movements of the muscles of the face, tic and spasm are the most frequent. These two terms are not infrequently confounded and one affection diagnosticated instead of the other. It will be brought out later that such a confusion leads to the most undesirable practical results. On the other hand, if the nature of the two diseases is properly recognized, considerable benefit may be derived from an appropriate treatment.

In order to understand pathological muscular movements, let us consider the physiology of the movements as they are observed in an infant and during the growth of the latter. An infant when touched or pinched, immediately removes the touched limb. If this is continued he moves other parts of the body, becomes agitated and screams, but he does not make an effort with the other limbs to remove the irritating object. The infant in such an experiment resembles a decapitated frog. When a drop of sulphuric acid is placed on one leg, immediately a movement of the same limb follows. When the irritation continues, the other leg will contract and the whole body become agitated. In the case of the decapitated frog as well as in the infant we deal purely with a spinal reflex. The infant is, therefore, speaking figuratively, a spinal being; it possesses only spinal reflexes. Here the motor reactions are accomplished without the aid of the

[1] Read November 6, 1912.

will and the cerebral cortex takes no part in them. The same remark is applicable to other movements or functions. Suction, for example, is also a plain reflex act; it is produced as soon as the lips come in contact with the nipple of the mamma. The brain is not in control. So it is with the respiration, as soon as the air strikes the mucous membrane of the respiratory passages. It is a pure bulbar reflex. As the physiological connection between the brain, medulla, and spinal cord is not yet established, the infant is unable to control or modify any of these movements or functions. Gradually, with the growth of the child, the physiological associations become established and the cerebrum gains control over movements and function. We then witness the voluntary movements of the child to remove from him any cause of irritation or pain. He learns how to execute complicated movements. If in an adult, we observe continuously automatic movements which are accomplished by him without the apparent interference of the will, such as walking, spitting, etc., it is because of a previous continuous repetition of the acts, acts which are done with one certain purpose. This so-called automatism could never be acquired without prolonged motor education which, on the other hand, could not be obtained without the aid of the will and always is a certain and precise direction.

These few physiological considerations lead to the following conclusions: Motor reactions may be of two kinds: one simple spinal reflex on which the will has no influence; the movements do not possess the character of the systematization or coördination. The others are coördinate and are executed for a certain functional purpose, but may be modified or controlled by the will.

To the first group belong spasm, to the second tic.

Tic of the Face. Tic is characterized by a sudden and abrupt contraction of one or of several muscles. As these are invariably the same and, as I said above, they are coördinate and are always executed for a certain functional purpose, the question naturally arises, why such a movement repeats itself and where is the purpose of it? The answer to this question will be found in the genesis of tic. A close examination will reveal the fact that

originally the movements of tic were made for a certain good reason and under the influence of the will. For example. A foreign body accidentally penetrated under the eyelid. Irritation is produced. The individual closes and opens the eyelids. He endeavors to get rid of it. Finally the body is removed. In normal conditions this is the end of the trouble. But certain individuals continue to reproduce the sensation of the foreign body. They will repeat the act of closing and opening the eye. Gradually he forgets the original trouble. He is satisfied with the act of repeating the eye movements. It becomes a habit and the movement becomes automatic. While at the beginning the movements were logical and reasonable, now they are superfluous. They constitute tic. It is evident that the original movement of tic is always an intentional act necessitated by special cause. Either a painful sensation or some other disturbance, in one particular spot produce a voluntary and coördinate act which, with the disappearance of the irritating factor, disappears in a normal individual, but persists in a person whose mental make-up is abnormal. The latter fearing the continuance of the original cause endeavors at first to make sure that it ceases to exist. He tries to find it and accomplish it, he has recourse to all sorts of gestures until he finds it. He finds satisfaction in experiencing again the original sensation. While the act is coordinate and systematic, nervetheless, it is unusual, illogical, because it is executed long after the original cause has disappeared; the act of closing and opening the eye is being produced in spite of the absence of the foreign body. It is evident that we deal here with an abnormal physical phenomenon and in a mentally unstable individual.

That a psychic element is predominant in tic, can be seen also from the singular satisfaction the individual experiences from indulging in tic movements after he makes an effort to arrest or prevent them for a certain time. Moreover, the patient's will can to a certain extent inhibit the tic movements, but unfortunately the efforts of his will are meagre and short in duration. A close examination of such individuals will reveal invariably a

history of instability, emotionality, affectivity, great variability of ideas, sometimes eccentricity or bizarre activities.

As a last characteristic physical diagnostic element of tic, we find that the muscular contraction in tic of the face do not correspond to a well-defined automatic distribution of a certain nerve supply. This is an important feature as it enables one to differentiate the disease from facial spasm.

Facial Spasm. Unlike the tic, the spasm does not present a reproduction of a purposeful physiological act. It consists of a motor reaction following an irritation of any part along the spinal reflex arc. It is strictly confined to the area of distribution of the facial nerve.

The muscular contractions in spasm may be of two kinds: either coarse or fascicular. At the time the spasm occurs in one half of the face, the muscles may contract either rapidly and produce the fascicular contractions, or more slowly and present the coarse variety. In the first there will be no wrinkles, in the latter cases deep wrinkles will be seen.

The movements in facial spasm are strictly confined to the area of distribution of the facial nerve, in contradistinction to tic which does not correspond to a well defined automatic distribution of a nerve. The muscles supplied by the upper facial nerve alone may be involved and then we observe a contraction of the musculus frontalis and of the orbicularies palpebrarum or else the lower facial nerve is irritated and the muscles of the lower part of the face are contracted, also those of the neck on the corresponding side. We have seen above that the twitching in tic is an exaggerated normal movement made originally with a certain object in view, such as the winking of an eye, etc. In spasm, on the contrary, the reason is illogical and without any reasonable expression; it does not resemble in the least any ordinary mimicry of the face.

The characteristic appearance of the face during a spasm can be described as follows: The forehead is wrinkled, the orbicularis palpebrarum closes the eye; there is a simultaneous contraction of the frontalis and orbicularis palpebrarum. This phenomenon

Babinski gave the name of paradoxical synergia. He calls it paradoxical, as normally when the frontalis is wrinkled the orbicularis palpebrarum cannot close the eye or when the orbicularis is contracted and closes the eye the frontalis cannot contract and raise the forehead. The angle of the mouth is drawn either laterally or elevated according to the mode of muscular contraction. In the fascicular spasm there is only a lateral deviation; in the coarse spasm there is a lateral and upward deviation of the angle of the mouth. The nose is curved in the direction of the spasm, the nasolabial fold on the affected side is deeper than on the normal side. The chin presents a depression on the side of the spasm. Another characteristic feature of spasm is its occurrence during sleep. The will has no inhibitory power, and therefore cannot arrest an attack. The opposite condition we find in tic. We have seen above that a psychic element plays a certain role in tic. The latter does not occur during sleep and can be controlled to a certain extent by cerebral interference. Besides, we have seen that the genesis of tic is really mental. (Gewohnheitskrankheit).

The pathogenesis of facial spasm is different from that of tic. It was mentioned above that a spasm is strictly confined to the area of distribution of the seventh nerve. A stimulation of any portion of this nerve or of its nucleus, or else of the sensory fibers of the fifth nerve will produce a spasm. The transmission of an irritation through the sensory fibers of the fifth nerve to the motor fibers of the seventh means establishment of a reflex arc. An irritation of the cornea or of the palpebral mucosa will produce a spasm of the orbicularis palpebrarum muscle. Bouchaud (*Revue Neurologique*, 1908, p. 901) observed a patient in whom a dental disorder developed a trigeminal neuralgia. Each attack of pain was followed by convulsive movements of the face. Here the seventh nerve was intact, but the fifth nerve was involved through a carious tooth. The stimulation was here transmitted from the fifth nerve to the muscles of the seventh nerve. An irritation therefore of any portion of the reflex arc is apt to produce facial spasm. In the majority of cases, however, an irritation of the

seventh alone is the cause of facial spasm. It can be demonstrated by the direct electrical stimulation of the seventh nerve. Moreover, it is quite common to observe a facial spasm following a facial palsy of long standing. In such cases there must be some localized irritating or destructive lesion of the seventh nerve. In A. Thomas' two cases (*Revue Neurologique*, 1907, p. 1273) there were neuromas directly on the facial nerve.

Treatment. In view of a different pathogenesis of the two affections studied here the treatment must logically differ. It was mentioned above how much tic was associated with the patient's cerebral functions, especially how it originated from the repeated mental operations. It stands to reason that the treatment of the acquired habit of convulsive movements should be directed toward the patient's psyche. He should be taught a mental discipline which should enable him to overcome by the efforts of his own will the ill directed habit. Two procedures, one following the other, can be utilized for that purpose. One can be called voluntary immobilization of the affected muscles; the other, movements of the immobilized muscles.

The first method consists of a systematic and regularly practised effort to immobilize the face. This training is done daily. At first for a brief period—only for a few seconds, two or three times a day. Later the duration of each individual exercise is increased, but this must be done very gradually. Haste in the treatment will meet with failure. If the patient is an adult, he can be taught how to exercise himself. I am in the habit to advise the patient to place himself before a mirror and observe his immobilization exercise. In the case of a child the procedure must be in charge of a trained person. When the person becomes proficient in immobilizing his face at command or at his own will the second method is then taken up. It consists of voluntary contractions of the affected muscles of the face. The patient is directed to produce slow, regular, and correct movements of the face. Here, again, the exercises must be very brief but frequently repeated. The séances must gradually increase in frequency and duration. The mirror again can be utilized to great advantage. In this

procedure particular slowness is very essential. In both methods patience and persistence are the necessary requisites. Satisfactory results follow only after prolonged and repeated trials. The treatment is undoubtedly tedious and frequently discouraging, but with perseverance and enthusiasm the patient will eventually overcome the distressing affection. As to drug treatment it is useless. It is well to bear in mind that tic usually occurs in individuals with peculiarly constituted nervous system. (See above.)

It is therefore advisable to strengthen the latter, to surround the patient with an existence, free from excitement and worry, to forbid stimulants, including tea and coffee, to advise him the proper hygiene and the proper mode of living. Electricity has been advocated for the treatment of tic by some authors, but in my experience it is a useless procedure.

The treatment of facial spasm in view of its different pathogenesis will be totally different from that of tic. We have seen above that in the majority of cases the spasm is caused by some irritation of the peripheral nerve trunk of the seventh nerve. It has been observed that injections of ethyl alcohol directly into the nerve removed the exceedingly distressing, although not painful, affection of the face. The method consists of injecting with an ordinary hypodermic syringe into the facial nerve in the stylomastoid foramen five minims of 80 per cent. solution of alcohol. The injection is done without a preliminary localized anesthetic. Anatomic knowledge of the exit of the nerve from the foramen is essential. As a point of departure, the end of the mastoid process can be taken. The foramen is situated in front and inward of the process. Striking the nerve with the needle is not an easy matter. Sometimes it requires many injections before the nerve is caught. In the case of the man twelve attempts were made vainly and only the thirteenth was successful. A successful injection means an immediate facial palsy and immediate disappearance of the spasm. If, after an injection, there is no deviation of the face we met with a failure. The paralysis is clearly of peripheral type; the upper as well as the lower branches of the facial nerve are involved. The injections are followed by

a certain amount of tenderness in the injected area which may last for weeks, but there is no actual suffering. The facial palsy resulting from a successful alcoholic injection is not permanent. It is advisable not to interfere with the artificially produced palsy for a period of four or six weeks, at the end of which time electrical stimulation of the palsied muscles may commence. It should be done like in any ordinary case of Bell's palsy, viz., daily stimulation of ten or fifteen minutes' duration at first with galvanism and later with faradism. The palsy usually lasts about three months.

My personal experience with eleven cases of alcoholic injections into the facial nerve for treatment of hemispasm have given me highly satisfactory results. Only in three cases had I a recurrence, but after one, two, and three years. In these the recurrent spasm has been by far less pronounced as before the injections of alcohol. All the three patients declined repeated treatment, and presently the twitching in every one of them is extremely slight. Three patients submitted themselves to a second injection and so far after a period of nine, sixteen, and twenty months respectively, there has been no recurrence. Five remaining patients had only one injection and so far there has been no recurrence. One of them has been under observation almost three years and no trace of spasm has been noticed yet.

The conclusion to which these observations naturally lead is that the procedure of alcoholic injections into the facial nerve is the most satisfactory of all therapeutic measures known at present. In spite of possible recurrences in some cases, one may have recourse to a second and even to a third injection. In some cases a single injection is apt to remove the twitching for a period of even three years, as it can be seen from my experience. The method is therefore to be recommended and urged. Besides the esthetic feature of the condition the affection is very distressing, and it may interfere with talking or masticating food, and if a simple procedure may give relief for a period of months also of years, it should be applied to every case of facial spasm.

It should be borne in mind that only a genuine facial spasm

can be treated with alcoholic injections, as the localized irritating factor is the seventh nerve. The treatment cannot certainly be applied to tic as the genesis of the disease is not in the facial nerve, but in the patient's psyche. It, therefore, must be treated by psychic measures but not by material agents. A rigorous differential diagnosis between facial spasm and tic must be made before the above described treatment is undertaken.

PHYSICAL EXAMINATION OF THE LIVER[1]

By JOSEPH SAILER, M.D.

The physical examination of the liver should reveal the following facts regarding it:

1. The position and contour of the upper limit of liver dulness and whether it moves with respiration.

2. The position of the lower border as ascertained either by percussion if it is above the costal margin or by palpation if it is below. This includes the recognition of the point where the liver passes beneath the costal margin to the left.

3. The character of the edge.

4. The consistency and character of its surface.

5. The palpability of the liver posteriorly. This involves bimanual palpation.

6. The motility of the liver. This may be of four kinds: (1) respiratory, (2) a gravity motility so that the liver changes its position with the posture of the patient, (3) passive motility, so that the liver can be moved in various directions by the palpating hand, (4) pulsation. The position of the upper border of liver dulness can be determined only by percussion. It is usually from one inch to one and one-half inches below the highest point reached by the liver in the thorax. The exact position of the liver during life can be determined only by means of the fluoroscope or a radiogram. It can also be determined at postmortem, most accurately probably in frozen sections. In the majority of cases the position of the liver is somewhat higher as determined

[1] Read October 2, 1912

by this method than it is during life, owing to the collapse of the lung which pulls up the diaphragm, and the sinking of the abdominal wall and therefore the diminution of resistance from below. Percussion is usually best performed by comparing the notes obtained in adjacent interspaces and then the notes obtained over adjacent ribs. If percussion is performed by striking alternately interspace and rib, there is apt to be some doubt regarding the exact point. If when a point is reached at which a dull or flat note is altered to a resonant note by deep inspiration, it may be assumed that the upper border of liver dulness is alternately above and below it.

Under certain circumstances it may be difficult or even impossible to determine the upper border of liver dulness at least in some portion of its circumference. It cannot, of course, be determined in pleural effusion and consolidation of the lung, nor in certain conditions in which the pleura is thickened. In any of these conditions the diagnosis is comparatively easy providing the lesion is of a pronounced character. It becomes difficult if the area of consolidation, the amount of exudate, or the extent of thickening is slight. Under these circumstances a sign first described, I believe, by von Leyden is of value. This is that in nearly all supradiaphragmatic conditions the upper border of dulness does not descend during inspiration. There is, however, an exception to this in the case of small effusion not sufficient to flatten the dome of the diaphragm. Under these circumstances the upper border of the fluid falls and rises within and expiration in the same manner as does the liver itself.

A patient was seen with Dr. Shellenberger, of Germantown, a robust, vigorous man who for three days had been unable to lie down and sleep on account of acute dyspnea. He had fever, temperature ranging to 102°, and a leukocytosis between 15,000 and 20,000, a practically normal heart, and both lungs were apparently normal, with the exception of a few crepitant rales at the right base. The upper border of apparent liver dulness posteriorly reached to the level of the ninth dorsal spine. It did not move with respiration. The lower border of pulmonary resonance on

the left reached the level of the eleventh dorsal spine. It moved with respiration. Anteriorly the liver dulness was at the sixth rib and moved slightly with respiration. On auscultation air could be heard passing in and out of the right lung and the chest expanded equally. In view of the absence of respiratory movement, the diagnosis of diaphragmatic pleurisy or empyema was made. An exploratory puncture withdrew pus, a subsequent operation by Dr. Stewart evacuated a considerable amount of seropurulent fluid contained in numerous cells between the lung and the diaphragm.

In another patient, a case of myocarditis in a man, aged fifty-six years, after the aspiration of a right-sided hydrothorax there was evidence of insufficient expansion of the lung as a result of flattening at the right apex and diminished expansion. Anteriorly there is impairment of resonance in the second interspace, which rapidly increases to flatness toward the base. The upper border of this impairment of resonance does not move with respiration. The breath sounds are faint, and in this area there are numerous crackling rales. Posteriorly the resonance extends to the tenth dorsal spine, but there is distinct respiratory movement. Posteriorly impairment of resonance begins at the sixth dorsal spine and shades into absolute flatness over the base of the right lung. Vocal resonance and tactile fremitus are quite distinct, and breath sounds, somewhat fainter than on the left side with prolongation of expiration, are distinctly heard. In this case there is evidently a thickening of the pleura densest toward the base and becoming less distinct toward the apex.

In a second case the condition is somewhat more obscure because the patient gives no history of any pulmonary disease, but beginning at the upper border of the fourth rib there is an area of dulness extending downward and merging with the liver dulness. It slopes obliquely downward and outward, and meets the liver dulness one inch to the right of the midclavicular line. It does not move with respiration. Over this area the breath sounds are faint, but nevertheless can be distinctly heard. The vocal resonance and tactile fremitus are diminished. The nature

of the lesion is obscure but from the normal condition of the lung and the diaphragm in the back it seems surely to be supra-diaphragmatic.

Difficulty may some time arise as a result of dislocation upward of the liver. In a patient upon my service at Blockley last winter the upper border of liver dulness was at the level of the third interspace, and moved with respiration. The lower border was at the sixth interspace and was movable. The only cause of this anomaly was the high position of the diaphragm on the right side, and it is, as far as my experience goes, a unique case.

In subdiaphragmatic conditions the position of the liver may be rather obscured by the presence of air, pus, and ascitic fluid between the surface of the liver and the under surface of the diaphragm. The greatest difficulty exists in the so-called subdiaphragmatic pyopneumothorax. The only satisfactory sign of this condition is the existence of a tympanitic area beneath the pulmonary resonance which moves with respiration. If within this tympanitic area movable dulness can be demonstrated or, provided the condition of the patient warrants it, and a succussion splash elicited, a diagnosis may be hazarded with some confidence. The history is often of great assistance. In a patient upon Dr. Tyson's service at the University Hospital two years ago, there was a history of severe hematemesis followed a few days later by intense epigastric pain, and then finally by the signs of suppuration somewhere in the body. In this case although air was subsequently demonstrated, no tympanitic area between the liver and pulmonary resonance could be demonstrated. Nevertheless the diagnosis of subdiaphragmatic pyopneumothorax was made, partly on the basis of movable dulness and the mobility of the upper border of liver dulness, chiefly, however, upon the history and symptoms. An exploratory puncture made by a surgeon failed to give either air or pus, and he refused therefore, to operate. Finally, the condition of the patient demanded intervention as the sole hope of prolonging his life, and an operation through the eighth interspace evacuated about a pint of intensely foul pus, and at the same time a considerable

amount of offensive gas escaped. Why the exploratory puncture had been futile was not clear.

If subdiaphragmatic pyopneumothorax is possible, and we know that it is, it is, of course, conceivable that subdiaphragmatic hydrothorax might occur; that is to say, in place of pus and air separating the upper surface of the liver from the inferior surface of the diaphragm an accumulation of serous fluid might accomplish the same results. I was not aware, however, that the condition existed until I met with a case which by some stretch of medical terms might be considered in this category. The patient was under the care of Dr. Hand, and by his courtesy I was able to study her. A woman, aged about fifty years, gave a history of having had severe hematemesis six months previously. Shortly after this she developed ascites, for which she was tapped, and subsequently hydrothorax. These two conditions recurred rather frequently, and tapping was done alternately in the abdomen and in the right thorax about every ten days or two weeks. The breath sounds could be heard as far on the right side as the level of the ninth dorsal spine posteriorly and the sixth rib anteriorly. Nevertheless the percussion note over the whole lower part of the right lung was impaired, and the day after examination several quarts of fluid were removed by a puncture in the seventh interspace in the posterior axillary line. It was noted at the time of this aspiration as well as subsequently that the abdomen was softer after the thorax was tapped. It seemed clear, however, from the presence of breath sounds, that the lung could not be floated upward by the fluid, and therefore the possibility of a subdiaphragmatic hydrothorax was suggested. This was readily confirmed by the *x*-ray, the diaphragm on the right side arching up into the chest wall. At the autopsy, which was performed some months later, a perfectly typical hobnailed liver was found and a huge cavity between its upper surface and the distended right diaphragm.

The position of the lower edge of the liver is best determined by palpation. This, of course, is not always possible. It often happens in a normal patient or in one whose liver for any reason

is drawn upward or diminished in size that the lower edge cannot, be felt even during deep inspiration. In such cases percussion must be used, but is apt to be misleading because the thin lower edge of the normal liver does not give a flat note, and the distance above the edge at which the note becomes flat varies in otherwise perfectly normal livers. The *x*-ray gives fairly satisfactory results, but they are only accurate if the orthodiagraph is used. No general rule for palpation can be given even if the lower edge of the liver is accessible. If the liver is enlarged and hard its edge can usually be traced by almost any method. Difficulty may arise because the wall of the abdomen is thick, and if this thickness is due to edema, then it may be quite impossible to determine the exact position. If there is merely a considerable layer of fat, the increased resistance over the liver may at least indicate the fact of its enlargement. If there is ascites and the fluid rises between the liver and the abdominal wall, sudden thrusting of the fingers downward will sometimes displace the fluid and allow the surface and even the edge of the liver to be plainly felt. If the wall of the abdomen is thin, perhaps the best method is to stroke gently from below upward. The edge of the liver is then readily felt, and can be traced around its whole extent. If the liver is simply ptosed, it is usually somewhat rotated downward and backward on an imaginary horizontal and transverse axis. The easiest way to detect its position is to employ deep stationary palpation at some point just below the supposed position of the edge of the liver. If now the patient takes a deep inspiration the liver can be felt as it comes down, at first above the fingers and then as it pushes up the skin it snaps gently beneath them. This method, however, is only available in the majority of cases for that part of the lower border which rises to the right of the right midclavicular line, and therefore does not determine positively the position of the left side of the liver. If in addition to the palpation anteriorly the other hand be placed in the right flank and strong lifting force employed, the character of the edge and consistency of the liver may be quite accurately ascertained.

Mistakes regarding the position of the lower edge of the liver may occur as the result of a considerable variety of conditions. The following case was observed on Dr. Musser's service at the Philadelphia Hospital when I was his assistant. The patient was an emaciated man, and it was supposed that the lower edge of the liver could be easily palpated, extending as far as the level of the umbilicus. At autopsy it was found, however, that the great omentum was greatly thickened, wedge-shaped, and firmly adherent to the lower edge of the liver holding the transverse colon beneath it. For some reason sections of this curious mass were never cut, or if cut were never reported, but it was presumably tubercular.

Various abdominal tumors may also give rise to confusion. Of these the most important is probably retroperitoneal sarcoma. In a case seen by the late Dr. Musser, the late Dr. J. D. Steele, and myself, which was subsequently reported by Dr. Steele, a huge mass occupying the epigastrium in a rather obese elderly lady strongly addicted to the use of alcohol, was supposed to be an enlarged liver. The surface was fairly smooth, but there is no record as to whether respiratory motility was tested or not. At any rate, at autopsy it was found to be a huge sarcoma springing from the retroperitoneal glands and lacking the usual nodular character of these tumors. The liver, of the hobnailed variety, was tucked away beneath the ribs in a position where it was quite inaccessible to palpation.

In a patient under my care suffering from chronic myocardial degeneration, a similar mass can be felt in the epigastrium that moves slightly with respiration. Its upper surface is quite granular, its lower edge rounded and almost circular. I supposed it to be an enlarged left lobe, but I feel very doubtful regarding its exact nature.

Carcinoma of the stomach, enlarged spleen, enlarged or ptosed kidney may possibly give rise to some confusion regarding their nature, or perhaps, and this is particularly true of a large kidney, render the delimitation of the liver practically impossible. An enlarged gall-bladder or a Riedel lobe are really parts of the

liver moving with it in respiration, but altering the contour of the lower edge.

It may be difficult or even impossible to palpate the lower edge of the liver in cases of extreme ascites or tympanites, and it is almost impossible to palpate it in case of pneumoperitoneum. I was recently called to see a patient who had been under my care for some years, suffering from hyperchlorhydria, but for a long period entirely without symptoms. Twenty-four hours previous to my summons he had suffered considerable epigastric discomfort. At half-past six in the afternoon the pain became excruciating, and he went into a state of collapse. I reached him about eight o'clock. The abdomen was distended, peristalsis markedly diminished, but there was no evidence of fluid in the flanks. The liver dulness could not be obtained, but it was replaced by a loud tympanitic note which extended to the resonant note of the lung. The respirations were shallow and frequent, the pulse that of collapse. As soon as possible an operation was performed, and on incision into the peritoneum a large amount of gas escaped. A ruptured duodenal ulcer was found.

The character of the surface of the liver can only be determined if the liver is enlarged or ptosed, and the wall of the abdomen thin or relaxed. It may be smooth, granular, or nodular. If there is much subcutaneous fat a smooth and nodular surface cannot be differentiated. If it can be definitely ascertained that it is smooth, it indicates either ptosis or enlargement due to some infiltration, degeneration, or congestion. If granular, almost without exception, it indicates some form of cirrhosis. A rough nodular surface found in a patient aged over forty years suggests secondary carcinoma. In my own experience I have never found it to be anything else, but there is no doubt that the same physical condition may be produced by syphilis, cirrhosis, abscess, or hydatic cysts. I have seen a number of times at autopsy livers which were extremely nodular as a result either of cirrhosis or syphilis, but in all cases they were small and could not have been palpated during life. It must be remembered that a syphilitic liver is frequently large, in one case of syphilitic cirrhosis, fairly large

nodules were found at autopsy, but they had not been recognized by palpation during life. This case was remarkable for the fact that although a Talma operation was performed and was successful to the extent that at the autopsy the collateral veins were found dilated to the thickness of ordinary fingers, the ascites continued to reaccumulate with undiminished rapidity.

Secondary carcinoma of the liver, as a rule, gives rise to an enlargement that is at first palpable in the epigastrium. It must be remembered that this palpable tumor may be the earliest indication that carcinoma exists. I have seen two patients in which it was discovered in men apparently in perfect health, and in both confirmed by exploratory operation, but in neither of these could the primary source be determined because autopsies were refused. In another case, after a prolonged irregular fever, a small lump, movable with respiration, quite hard, and moderately tender, appeared beneath the left border of the ribs in the extension of the parasternal line. Considerable uncertainty was felt for a long time. The high leukocytosis and the irregular fever suggested an abscess, but at the exploratory operation only numerous nodules were found. One of these nodules was excised, and in addition to the histological study a careful search for parasites, particularly tubercle bacilli, was made, but the results were negative.

Bimanual palpation of the liver is of importance in determining its thickness. The Riegel lobe is usually quite thin, not more than one-half to one inch in thickness. In cases of hepatosis the liver appears to rotate so that although the lower edge is considerably beneath the costal margin, the substance of the liver is not very thick, and the posterior hand only comes in contact with it when firm pressure is made from above. On the other hand, in cases of diffuse enlargement of the liver, particularly in passive congestion and secondary carcinoma, the massive liver fills the right flank and the palpating hands are separated by the thickness of the body.

The liver, as a rule, shows marked respiratory motility. This is so constant that it is reasonable to infer that any fixed organ

in the upper part of the abdomen is not hepatic but is more likely to be either a retroperitoneal sarcoma or an abdominal aneurysm. I have seen but one case of obliterative perihepatitis, and this was discovered at operation only because the distention of the veins in the wall of the stomach led to a careful search for the cause of the venous congestion. In this case it could readily be seen that the liver moved with respiration, but I regret that my interest in the operation itself led me to fail to make any attempt to determine whether the movements were restricted. Indeed, it is difficult to understand how such restriction could occur unless there existed at the same time some condition binding the diaphragm to the parietal pleura.

A passively movable liver may occur. The fact that the liver can be so widely separated from the diaphragm as occurs in subdiaphragmatic abscess, pneumo- and hydrothorax is sufficient indication that the ligaments are capable of considerable extension. The degree of passive motility that can be obtained in cases of ptosis of the liver is considerable. It is, however, limited to a moderate excursion, and can be recognized by placing the hand on either side of the liver or in front and back and pushing it from one side to the other. Passive motility is not of great significance excepting in distinguishing the liver from some fixed tumor. There is a form of passive motility that occasionally is of some service. It is obtained by pulling or pushing some structure to which the liver may or may not be attached, and in this manner determining whether they are connected or not. Only in one instance have I been able to employ this with a positive result, a case of rounded tumor situated in the right flank, palpable bimanually, which, when it was forced downward, dragged the liver with it. The patient was a girl, aged fourteen years, slightly jaundiced. At the operation what was supposed to be a congenital biliary cyst in the mesentery was found.

Movement of the liver may produce movement in some structure attached to it. This is particularly valuable in cases of distended gall-bladder. The nature of these tumors is usually readily determined by other means, but occasionally when the

liver is pushed from side to side their movement with it is of considerable support to the diagnosis.

There only remains to be mentioned pulsation of the liver. This occurs in three ways, either the pulsation is transmitted from the aorta; this rarely occurs, except in the left lobe; or there is ventricular pulsation as a result of tricuspid insufficiency. This is very common, and can be felt anteriorly, and also posteriorly beneath the twelfth rib and bimanually. Posterior pulsation in the right flank is pathognomonic of liver pulsation. It occurs in no other condition. Finally there is auricular pulsation of the liver discovered only by the use of graphic methods, originally by McKenzie. This was at first supposed to be due to tricuspid stenosis and a consequent hypertrophy of the right auricle, but it is now known that it occurs in other conditions. In the only case that I have ever observed it was associated with an extreme degree of mitral stenosis and the tricuspid valve was freely patulous.

DISCUSSION.

Dr. Herman B. Allyn.—Dr. Sailer has referred to some of the difficulties encountered in determining the cause of enlargements of the liver. I am reminded of two cases seen last winter: One patient was a boy who had a large liver as the result of cardiac disease. This boy developed late in the disease a fluctuating tumor on the surface of the liver at the usual site of the gall-bladder. It was supposed to be an enlarged gall-bladder, but later there appeared also other nodules about the size of marbles. The mass was distinctly fluctuating and, inasmuch as the blood gave a positive Wassermann, we looked upon it as possibly softened gummas. But at autopsy we found cysts beneath the serous coat of the liver. I never before saw nodules of this size and due to this cause. The specimens were shown before the Pathological Society last winter. The other case was that of a woman with a secondary sarcomatous mass developing beneath the left border of the liver which moved freely with respiration. It was found to be a mass connected with the omentum and not attached to the liver.

A STUDY OF EMPYEMA, WITH SPECIAL REFERENCE TO THE FEASIBILITY AND IMPORTANCE OF DEPENDENT DRAINAGE.[1]

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The large number of operations which have been done in recent years has added much to our knowledge of the pathological changes which have taken place and the mechanical conditions which have arisen in connection with the various diseased organs or tissues on account of which the operations have been performed. Our opportunities for the study of a pathological condition are much improved when during the operation it becomes necessary to remove the diseased organ or tissue, as in appendectomies, gastrectomies for ulcers or carcinoma, or in operations for the removal of other tumors. Even when nothing is removed a free exposure of the affected part is often obtained, so that practical observations can be made from sight and touch. Yet from the large number of operations which have been done for empyema we have learned very little in this way. The opening made in the chest wall is too small for inspection of the diseased parts within, in the presence of the escaping pus, and the finger is too short for palpation. Our efforts, of necessity, have been confined chiefly to bacteriological examinations of the pus evacuated and to a study of the relation of the pus collection to the preceding infectious condition of the lung, as pneumonia or phthisis. Robinson[2] says that the bacteriology is of interest in prognosis, but of

[1] Read November 6, 1912.

[2] Boston Med. and Surg. Jour., 1910, clxiii, 561.

no significance as regard treatment. All cases should be recognized promptly and drained freely, reëxpansion of the lung being the main purpose from the start. Most surgeons will probably agree with these statements.

In a previous paper[1] on empyema I employed an illustration of a formalin-hardened cadaver specimen, from the department of applied anatomy of the University of Pennsylvania, of an empyema from which the pus had not been evacuated during life. The possession of this specimen is the result of an accident, it having been selected for injection with formalin as one in which the normal relations and conditions of the abdominal and and thoracic organs should be found, and I used it for the first time to illustrate how easily a large empyema could be overlooked on inspection. I have come to the conclusion that it was worthy of a more careful study and that it might yield valuable practical information concerning empyema in general. When making a study of conditions in the dead body we usually obtain our material from the dissecting or postmortem room. This kind of a specimen could hardly be obtained from either. It must be specially prepared, and one must know that the unopened empyema is in a body before he prepares it, unless, as in this case, he happens to find it by accident after he has injected the body with formalin. The number of empyemas that find their way into the postmorten room unrecognized and unopened is probably very small, and those that do are usually so mutilated before the empyema is recognized that the preparation of a formalin specimen of this kind would be impossible. The first incision and manipulations necessary to expose the empyema would render the injection of the formalin ineffective, and would distort the relations of the soft structures to each other. In the dissecting room the conditions would be similar or worse for the preservation of a similar specimen. If an opening had been made during life, such changes would have taken place that would have rendered the specimen less valuable, because we are most interested clinic-

[1] Internat. Clinics, 1906; iii, 150.

ally in the empyema before the pus has been evacuated. Fig. 1 shows that there existed here the large common and typical

FIG. 1.—Formalin hardened cadaver specimen of an empyema which has been completely circumscribed by firm adhesions above between the lung and chest wall. A small portion of the lung, narrower than appears in the illustration, remained adherent to the diaphragm. The costophrenic sinus is much wider than normal from the pressure of the pus, while on the opposite side it is almost obliterated. The heart pushed to the left looks larger than it actually is because abnormally uncovered by the left lung which is crowded posteriorly and to the left. The placing of the drainage opening at the bottom of the normal pleural cavity (see Fig. 2) would have been easy and safe in this case, as it proved to be in the three cases in which it was so placed. The large intestine is much distended and the liver distorted and depressed by the pressure of the pus.

empyema, that giving marked and superficial dulness extending from the bottom of the pleural cavity a variable distance upward

and merging below on the right side with the liver dulness. The displacement of the heart and liver are also typical.

A bacteriological examination of the pus debris remaining in the specimen was made by Dr. Rivas, of the University of Pennsylvania. He found in it leukocytes, lymphocytes, two kinds of bacilli, and a diplococcus. One of the bacilli was acid-fast, and suggested the tubercle bacillus. This was confirmed by a longitudinal section of the affected lung. The specimen had been lying in a strong formalin solution for about eight years, so that the pus debris from which Dr. Rivas made his examination had undergone such change as not to permit an exact bacteriological report. He concluded that the pus had been the result of a mixed infection. The most striking and important feature in connection with the specimen, in my opinion, is that the empyemic cavity, although a large one, is distinctly and firmly circumscribed below by the junction of the diaphragm and chest wall and above by adhesions between the lung and chest wall, so intimate and continuous that the line of cleavage between the two cannot be seen. I have seen a formalin preparation of a tuberculous empyema in which there were no adhesions between the two pleural layers, the lung lying completely collapsed against the spine. The probability is that in the latter there had developed a communication between a bronchus and the pleural cavity, resulting in a pneumothorax in the presence of a slight tendency toward adhesion formation. In my specimen there could be found no evidence of such a bronchial communication after a careful search of all surfaces presenting on the empyema, which was aided by a removal of a longitudinal section of the lung, about 3 inches wide, and extending the whole length of the lung. The firm and extensive adhesions sharply circumscribing the empyemic cavity were probably due to absence of a bronchial communication, and the fact that there was added to the tuberculous a pyogenic infection; the latter having a greater tendency toward adhesion formation than the former. This does not mean that tuberculous infection cannot produce the condition exhibited by this specimen. There is much vagueness in the literature con-

cerning the existence of and the part played by adhesions in empyema. Many writers ignore their existence; some refer to bands of adhesions; while others seem to think their existence is largely a matter of chance. If they developed extensively in the presence of a mixed tuberculous and pyogenic infection, as in this case, I believe they are almost certain to occur in the acute pyogenic infections, including the pneumococcic, in which pneumothorax is rare. Indeed, the burden of proof should not be upon him who says that such adhesion formation is the rule, but upon him who says that it is not.

The empyema in this specimen extended from the bottom of the normal pleural cavity upward, anteriorly at one point as high as the second rib and posteriorly in a rather broad channel as high as the apex. It was one continuous cavity and no bands of adhesions crossed it. It was thoroughly walled off on all sides: above by close and firm adhesion between the lung and chest wall, and below by the union of the diaphragm and chest wall. I have based my study of empyema chiefly upon the character of the adhesion formation in this specimen, and upon the assumption that it is the rule in acute empyema. Many of the accepted teachings in connection with empyema, according to my judgment, have either taken no account of this feature or have not given it sufficient recognition. I have drawn some inferences from my study that I look upon more as suggestions than conclusions. The main definite conclusion I would draw is that dependent drainage in empyema, which calls for an opening as low as the eleventh rib or to the top of the twelfth rib in most cases, is not only feasible but comparatively easy and safe.

This massive type of empyema is frequently referred to as the unencysted or general, which implies that the pus is free to flow into all parts of the affected pleural cavity. The specimen strongly contradicts this conception, and shows, in my opinion, that practically all acute empyemas are encystèd and localized by surrounding more or less firm adhesions, as in an appendicular or other abdominal abscess, or the small so-called localized

empyema. The latter is localized, but probably no more so than the large one.

Morison[1] says that the collecting effusion is first observed in the region of the fifth interspace, that is, diagonally farthest from the fixed point of the lung at its root, and that the reëxpanding lung last touches the chest wall in the same region. For this reason he makes this the point of election for the drainage opening. According to my experience most empyemas extend from the bottom of the pleural cavity, as in this specimen, a variable distance upward. I believe that the fluid begins to collect not about the fifth rib, but at the bottom of the normal cavity. Donaldson[2] says that "the fluid products in pleurisy gravitate by their weight to the lowest portion of the cavity of the pleura and then gradually rise." The last part of the lung to reëxpand is probably that about the drainage opening, at whatever level it may be, where the external atmospheric pressure acts longest and to the best advantage. Considerations based upon this specimen suggest the following conception as to the conditions developing in and about an empyema. Most pneumonias affect the base of the lung, therefore the resulting pleural effusion should begin at the base of the pleural cavity. But let us suppose that the infection involves a higher portion of the lung and reaches the pleura on the corresponding portion of the external surface of the lung. If adhesions develop between the visceral and parietal pleuræ, here, there can be no empyema because there is no space in which the fluid can collect. If no adhesions develop the excess of fluid which results from the pleurisy falls immediately to the bottom of the pleural cavity and prevents adhesions between the surfaces over which it continues to fall. It should collect first in the costophrenic sinus, pushing the lung here away from the chest wall and diaphragm and the latter two from each other, as in the specimen. I would again agree with Robinson when he says that he is "not in sympathy with Fowler, who says that adhesions may occur in the costophrenic angle, raising the level

[1] Trans. Med. Chir. Soc., Edinburgh, 1907 to 1908, vol. xxvii.
[2] Pepper's System of Medicine, iii, 487.

of the bottom of the cavity so that the drainage need not be at the lowest point." Such adhesions might develop after a drainage opening had been made, but are not likely before. The accumulating fluid pushes the serous surfaces apart, but sooner or later where they are in contact above the pus collection and around the whole lung the approximated surfaces develop adhesions, weak at first but gradually growing firmer. In this way the pus becomes walled off all around by adhesions between the lung and chest wall, and probably some part of the diaphragm. This walling off is much more likely than with the average pus collection in the abdominal cavity, because in the pleural cavity the pus is less likely, from the nature of the cavity and the usual position of the patient, to move about. For the same reason a pelvic infection is less likely to give rise to a general peritonitis than one in the appendix or gall-bladder region. The cadaver specimen shows clearly that such a walling off occurred in this case, and that it usually occurs in empyema is strongly indicated by the fact that when a patient is changed from the sitting to the recumbent position there is little or no variation in the upper line of dulness. Movable dulness is probably rare in empyema, except perhaps in the early stages, before the adhesions have become strong enough to withstand the pressure of the pus under the influence of gravity. Donaldson says: "Once formed, these effusions (of pleurisy) are but slightly movable and but little displaced by the varying positions of the patients, unless the quantity be very great and no adhesions or bands have been made." He also says, "The serous transudations of hydrothorax always occupy the most dependent portion of the cavity." In other words, the fluid in the latter moves about under the influence of gravity. In an empyema the weight of the pus might cause a slight change in its position by pressure against the more or less soft lung. We know that the upper line of dulness in the sitting position is not straight but curved, the curve varying more or less, and that it does not change materially when the patient is placed in the recumbent position. The varying curve of this line is probably dependent upon the position occupied by the

patient during the developemnt of the limiting adhesions. The absence of movable dulness as determined by percussion before and after a change in the position of the patient should be a sufficient guarantee that the pus has become circumscribed by adhesions in any given case.

The adhesions probably developed early and became stronger as the quantity of pus and therefore its upward pressure increased, so that they held the adherent surfaces of the lung to the chest wall while the under surface of the lung removed from the chest wall was being pushed upward, the pus therefore reaching its highest level a variable distance from the chest wall. Above this level, especially when the pus collection is large, the percussion note is often hypertympanitic—Skoda's resonance—particularly in the first and second interspaces anteriorly. In the formalin specimen the pus at one point reached as high as the second rib, so that in large collections the first and second ribs or interspaces anteriorly cover about all of the lung above the pus that comes as close to the skin surface. Posteriorly and above the first rib anteriorly it is covered by a much thicker layer of muscles and other tissues. The inference to be drawn is that the lung is more tympanitic anteriorly in the first and second interspaces because it is nearest to the surface here. Skoda's resonance is explained by a retraction and relaxation of the lung from its elasticity and the upward pressure of the pus. This implies that the lung is being floated upward by the pus causing it to become relaxed. It has seemed to me that retracted and relaxed lung must be more or less functionless according to the degree of collapse, resulting from the relaxation. If its air cells are full of air and functionating it can hardly be collapsed. The support which the lung obtains from its firm attachment to the chest wall in its whole circumference must aid it in resisting the upward pressure of the pus and in keeping its air channels open for respiration. The lung is not floating in the ordinary sense on the fluid pus, and therefore largely collapsed and functionless, but is probably trying to maintain its function with the aid of the extensive firm adhesions, the atmospheric

pressure exerted through the trachea, and with the aid of an increase in the capacity of the thorax produced by the auxiliary muscles of respiration. The portion of the lung tissue caught between this air pressure above and the pus pressure below is probably compressed and condensed, not relaxed. In the normal lung conditions the tidal air is only about one-third of the air contained in the lung. Necessity may now increase the proportion of the tidal air in the functionating portion of the lung, so that the hypertympany may be the result of overdistended, not relaxed lung. Auer and Meltzer[1] say that the actual necessity of respiratory function for the mere maintenance of life amounts only to a small fraction of the normal respiration, and that the extent of the changes of gases occurring in normal respiration with a closed thoracic cavity exceeds greatly the need for the maintenance of life, since normal respiration is provided with an abundance of factors of safety. Necessity demands in an empyema that any still functionating portion of the lung shall work to its full capacity. It is generally agreed that the lung on the sound side is doing compensatory work, why not any sound lung on the affected side? That it probably does so is strongly indicated by the fact that a patient can live with a large empyema on each side, and the fact that he can live under these circumstances, indicates that the pressure of the large collection of pus on each lung is being largely offset by the extensive firm adhesions in the whole circumference of each lung. These began to develop early and resisted with increasing firmness the increasing presssure.

If a considerable portion of the lung is so functionating then it follows that this portion is occupying about its normal proportion of the thoracic cavity, and the portion which formerly occupied the space now represented by the empyemic cavity must be more or less compressed and condensed below the functionating portion. The adhesions were developed in the early stages between surfaces about in their normal relations to each

[1] Jour. Exper. Med., 1911, xiv, 569.

other, and they probably continued to hold these surfaces together against the pressure developing later from the accumulating pus. This means, in my opinion, that the adhesions will not seriously hinder the reëxpansion of the lung after the pus has been evacuated, and that the cavity is to be filled by reëxpansion of that portion of the lung which presents upon or projects into the cavity. The adherent surface of the lung is about where it should be when the lung is fully expanded, and therefore the adhesions should not be broken up to favor reëxpansion of the lung.

Elsberg[1] reported some interesting facts concerning pneumothorax, observed in experimental work on dogs. Many of the dogs either died suddenly as soon as an opening was made into the pleural cavity, or a violent expiratory dyspnea ensued, soon followed by rupture of the mediastinal septum, double pneumothorax, and death. While most of the dogs stood well, a small opening in the chest wall, sudden heart stoppage or dyspnea, and death followed in some instances. The larger the opening, however, the more likely is the occurrence of serious interference with the respiration. In almost all the animals in which the size of the opening approached or exceeded that of the diameter of the trachea, when the dog was lying on its back, dyspnea and death followed. It made no difference in what part of the chest wall the opening was made. The cause of the rapid death seems to be the development of the double pneumothorax. If a dog will die of a total double pneumothorax a man should. But it is now known that a double empyema may be opened on both sides at the same operation and the patient live. Hellin,[2] after a study of 113 cases collected from the literature and 1 of his own, makes some very interesting statements in this connection. He says: "The question of a double empyema is closely related to that of a double pneumothorax. *A priori*, one would expect that a simultaneous opening of both pleural cavities would prove fatal. At first it was considered best to open the second pleural cavity after the first had been closed. (An interval of one hundred

[1] New York Med. Rec., 1908, lxxiii, 846.

[2] Berlin. klin. Wocb., 1905, xlii, 1415.

and fifty days was allowed to elapse in one case.) But compelled because of the severity of the constitutional condition, it was gradually found that the interval between the opening of the two cavities could be shortened until both were opened at the same operation. The skeptic can convince himself that the classical theory born of a fear of a double pneumothorax is unfounded, since the patients do not cease to breathe in spite of the two opened pleural cavities." Hellin in his case found that the cyanosis occurring during anesthesia disappeared on the opening of the pleural cavities. In experiments on dogs he noted that some of the animals survived even after the collapse of both lungs if the opening in the first cavity was closed. Auer and Meltzer showed that while from the opening of the pleural cavity in the dog, collapse of the lungs, dyspnea, and death occur, if the opening were closed after the collapse of the lungs had occurred, the dog would continue to live. Only an open pneumothorax causes dyspnea. When the cavity was closed, even though the lungs have recently collapsed, an inspiration will cause some distention of the lungs and an inspiration of air into them. An expiration will cause a compression of the lungs and a removal of the carbon dioxide. Hellin[1] inferred from his studies that a total double pneumothorax was not necessarily fatal, but that as in caissons, it is chiefly in the suddeness of the change of pressure that the danger lies. But he assumed also that simultaneous opening on both sides of a double empyema gave rise to a total pneumothorax on both sides. Fabrikant[2] asks if we may not hope that under these circumstances pleural adhesions will prevent collapse of both lungs. The formalin cadaver specimen gives an affirmative answer to this question. It is possible that the adhesions in the early stages may break down at some point and admit air to any open part of the pleural cavity above; but this would probably be very rare, if it occurred at all. The empyema specimen which represents probably the great majority of this massive type shows clearly why a patient can live after

[1] Archiv. f. Klin, 1907, lxxxii, 866. [2] Deutsch. Zeitschr. f. Chir., 1911, cviii, 584.

the simultaneous opening of a double empyema on both sides. The air is admitted not into the whole pleural cavity of each side, but into only the strongly walled off pus cavities, which are now extrapleural in the sense that an appendiceal abscess becomes extraperitoneal. The atmospheric pressure can now exert its effect only upon the surface of the lung presenting on the empyema which is buttressed against a threatening collapse by the strong adhesions which before the opening was made protected it against the severe pus pressure. The atmospheric pressure is always the same, but the pus pressure will probably vary with the quantity of pus present, so that the immediate effect of the drainage opening on the lung will probably depend upon the difference in the two pressures one way or the other. It is my impression that I have seen patients breathe better after the operation when they are resting quietly in bed, and that I have seen cases in which the breathing was not as good.

The fear is sometimes expressed that the rapid evacuation of the pus following the usual opening made by the resection of a small portion of a rib will be accompanied by a dangerous change of pressure on the surrounding displaced organs, especially the heart. Hellin feared this and advised that all empyemas, unilateral as well as bilateral, be aspirated immediately before operation or one or two days before, in order that a too sudden change of pressure may not take place. I have seen a fairly large number of empyemas opened, but I have never seen such a danger threaten, and I believe that it is very rare. With each outflow of pus from the opening on expiration there immediately follows an inrush of air on inspiration to take the place of the pus evacuated, until when the pus is all evacuated its place has been taken by air at a pressure of about 15 pounds to the square inch. There is therefore probably no great change of pressure, and certainly not a sudden one. The ominous loud noise made by the air rushing in through the opening with each inspiration is in reality an indication not of danger, but of safety. As already stated, Hellin observed in his case of double empyema that the cyanosis occurring during narcosis, disappeared after the empyemas had been opened on both sides.

I have operated upon three and possibly four of the so-called localized or encysted empyemas. Since I believe that the usual large variety is in practically all cases circumscribed by adhesions, I believe that these names do not properly distinguish the small ones. The status of these cases has not yet been decided. I would suggest that they are practically all interlobar or between the diaphragm and under surface of the lung, and that that is the reason for their small size, the pus becoming walled off by adhesions before it can get out of the fissure or the space between the diaphragm and under surface of the lung.

In connection with these small empyemas, Musser[1] emphasized the importance of firm and deep pressure in the intercostal spaces along the fissures and along the margins of the diaphragm or upper borders of the liver, to develop tenderness indicating the presence of small empyemas between the lobes of the lung or between the lung and diaphragm

Murphy[2] says that many of the cases diagnosticated as abscesses of the lung are actually the result of empyemas communicating with a bronchus. Those diagnosticated as lung abscesses are very likely small and covered by more or less lung tissue, that is, they are interlobar, The pus breaks into the lung and ultimately finds its way into the air passages because it cannot well extend in any other direction. Depending upon the depth in the fissure at which it develops, the pus may or may not extend along the fissure and come to lie directly under the chest wall when a small area of absolute dulness could be detected. It may fail to do so because of its depth, when it will be covered by a layer of lung tissue, the dulness be relative, and air sounds be detected over it. The fissure is so oblique that it would be practically impossible to evacuate some deep collections by finding the fissure through an opening in the chest wall, and usually in such cases it is necessary to reach the pus by going through the lung tissue covering it, first shutting off the pleural cavity if necessary by suturing the lung to the chest wall in the circumference of the opening in

[1] Jour. Amer. Med. Assoc., January 5, 1907, xlviii, 24.
[2] Ibid., 1898, xxxi, 281.

the wall. The presence of sufficient adhesions here would render this unnecessary. When the pus collection is in the outer portion of the fissure directly under the chest wall the lung in the circumference of the empyema will be firmly adherent to the wall. In my 4 cases, I had 3 of the superficial variety and 1 of the deep. Small empyemas may develop between the base of the lung and the diaphragm, as already stated, but I have had no experience with this variety. It is said that these small empyemas frequently develop on the external surface of the lung between it and the chest wall. If they do it probably will be in an adherent portion inclosing a non-adherent area. The probability is, however, that such a condition is rare, and if a careful examination is made it is likely that in these cases the pus collection is close to some part of one of the main fissures. Robinson believes that most of these cases are peripheral abscesses of the lung. He refers to 2 cases which he saw at autopsy and 3 upon which he operated, in all of which the evidence as he interpreted it pointed to their having originated in abscesses of the lung. This evidence consisted in the fact that he found lung parenchyma devoid of pleura on the pulmonary side of the cavity. He expected to find the inner wall of the empyema made up of visceral pleura. I believe with Robinson that the pus will not often break through the pleura into the lung substance in the usual large empyema, that between the parietal and visceral pleuræ. As already stated, I believe that an interlobar empyema remains small because the pus becomes circumscribed soon from the fact that it cannot easily spread itself out in the fissure. Gravity probably has little effect on it, and the movement of the apposed pleural surfaces normally is probably slight. As soon as it becomes walled off the pus can increase in quantity only at the expense of the lung which surrounds it. When it can no longer make room for itself by pushing the lung aside it will destroy it as it will destroy any other tissue which confines it, even bone. The pleura will probably first become necrotic and then the lung tissue and the process will continue until the pus finds an exit or kills the patient. Sometimes it will burrow externally through an inter-

space between the ribs. The lung is the weaker tissue, however, and therefore evacuation through the trachea is the more common. We should not expect to find the pleura intact on the visceral side if the empyema has existed long enough to destroy it, and in many cases it probably has, by the time the empyema is opened, because this variety is usually the most difficult to detect.

The main purpose of any general study of empyema should be to improve our methods of treatment. In recent years we have had a number of valuable contributions looking toward this end. The main discussion seems to centre about the question of the method of drainage, and the consideration which has received most attention is that of the influence of the atmospheric pressure admitted through the drainage opening. Most cases get well sooner or later from drainage in the usual way, but in a considerable number the closure of the opening and cavity is much prolonged, or they may not close at all. Murphy[1] says that before the surgeon makes an opening into an empyema he should be reasonably certain that it will close later. He has discarded operation in all cases in which there is not an external opening or a communication with a bronchus. In other words, if air has not already gained entrance into the empyemic cavity through an external or an internal opening he will keep it out. He aspirates the pus and injects into the cavity a few drams or ounces of a 2 per cent. formaldehyde and glycerin solution, repeating the procedure at intervals of a few weeks as the effusion re-collects, until it has entirely disappeared. The fact that Murphy employs it in all cases, with the exceptions mentioned, attests its value and alone makes it a method which will be widely tested when it has been fully placed before the profession. As yet he has spoken of it only briefly in the discussion of papers by others. The formalin is said to render the cavity sterile and to favor the absorption of the exudate. Tennant[2] tried the method, but in one case it failed him and a resection of a rib was

[1] Section on Surgery of the American Medical Association, 1909, p. 921.
[2] Jour. Amer. Med. Assoc., August 15, 1908, p. 572.

done. It is the ideal method for combating the ill effects of atmospheric pressure on the external surface of the lung, and if it comes into general use there will be fewer cases calling for the severe Estlander or Schede operations. But it has not yet received general approval, and the prevailing treatment is that with drainage.

Many devices and methods of obtaining drainage with the exclusion of air from the cavity have been offered, and recently von Eberts[1] and Robinson have added valuable contributions on this phase of the subject. Except for a brief attempt in one of my cases, which was soon discontinued, I have not as yet had any experience with these methods. It seems to me that before we can determine, with satisfaction, the relative value of suction or siphon methods of drainage we must first know approximately the limit of rapidity with which an empyemic cavity may be permitted to become obliterated. We are dealing with an abscess the walls of which are infected, and few surgeons will risk efforts at disinfection by irrigation. The rule is to permit the cleansing to be done by the natural processes after effective drainage has been instituted. In most cases in which drainage is employed in the usual way, without the use of suction, the cavity cleanses itself spontaneously and is obliterated by the expansion of the lung, sooner or later. Fraley[2] reported a study of 500 cases of pleurisy treated at the Pennsylvania Hospital. About one-fifth of them were empyemas. No case was cured in less than four weeks. The average was ninety days, in which column were found one-third of the cases. All but 6 were cured in four months or less, and these were severely complicated. The shortest duration of convalescence obtained by Robinson with his method of rib trephining and suction drainage was four weeks. although in the one fatal case, in which death occurred two weeks after operation, the lung was fully expanded as shown at autopsy. We might argue with some force, therefore, that about four weeks are

[1] Von Eberts and Hall, Montreal Med. Jour., June, 1910, 513; Annals of Surg., 1910, lii, 502; Idid., 1911, liv, 59; Jour. Amer. Med. Assoc., 1912, lix, 264.

[2] Amer. Jour. Med. Sci., May, 1907.

necessary, under the most favorable conditions, for the disinfection necessary before the cavity and drainage opening can close permanently. But the conditions vary. The degree of firmness with which the lung has become fixed in its compressed condition will affect the rapidity of expansion. The character of the infection will affect the rapidity of spontaneous cleansing, as will the presence of necrotic tissue in the wall of the empyema. According to Schädler, quoted by Hellin, the average duration of a unilateral empyema was fourteen and one-half weeks, in children nine weeks. Hellin says that the death rate in 1335 cases of unilateral empyema was 22.4 per cent. or 299 deaths. The mortality in the double empyemas was 30.1 per cent. Robinson evidently made a careful study of other methods of suction drainage than his own, and of them he says: "It is well known to those who have performed rib resection and introduced tubes protected by rubber collars, rubber dam, adhesive plaster, lanolin, zinc ointment, gutta percha, cement, and other agents that leakage is of frequent occurrence and that at best it can only be prevented by the most painstaking and continuous efforts." In his discussion of this subject at the meeting of the American Medical Association in June, 1912,[1] and at the meeting of the Pennsylvania State Medical Society, September 26, 1912, Robinson said that he now confines the use of suction drainage to only the most carefully selected cases.

It is an interesting fact that while the atmospheric pressure soon after the usual drainage opening is made is probably as great in the empyemic cavity as that exerted within the lung through the trachea, because the drainage opening is usually as large or larger in diameter than that of the trachea or larynx, in most cases the lung will expand later and obliterate the cavity. This is the more interesting when we take into consideration the fact that to the external pressure must be added the resistance of the normal elasticity of the lung and that of the inflammatory thickening of the inflamed pleura and adjacent lung tissue. In many cases this result will be accomplished in about four weeks

[1] Jour. Amer. Med. Assoc., 1912, lix, 272.

without the employment of negative tension from suction drainage. Yet it is obvious that the lung can be expanded only by the atmospheric pressure coming through the trachea. The internal pressure in some way must gain on the external pressure. I have in several cases introduced my finger through the drainage opening at the time of the operation to find that I could not feel lung tissue or only a small portion of it. It was being held beyond the reach of the finger by the external atmospheric pressure exerted through the drainage opening. In 1 or 2 cases I have introduced the finger at intervals later to find that the surface of the lung could be left to be approaching the opening. To my mind there is only one satisfactory explanation for it. The atmospheric pressure within the empyemic cavity must, in some way, be gradually diminished, so that the internal pressure can act to better advantage in expanding the lung. Otherwise the lung never could expand. In other words, the effect of the drainage opening usually employed is to provide more or less imperfectly, a negative tension within the empyemic cavity. We are all familiar with the sound made by the air which passes through the opening on inspiration during the after-treatment even in the presence of a pus-soaked dressing. I observed in one of my cases that this sound ceased soon after the operation, perhaps in a few days, and in that case the cavity and sinus were closed in five weeks. I saw at the Philadelphia Hospital some years ago an empyema patient who had been operated on about two and a half years before. The drainage opening was more than an inch wide at that time. There was no sound in that case from the passage of air in or out, probably because the opening was so large. I could look through it into the cavity, and when I saw the case again a year later, as far as I could judge, no progress had been made toward the obliteration of the cavity and a cure. I have observed that the sinus is not long in closing after the sound made by the inrush of air on inspiration has ceased. Whether the sound ceases because the cavity has almost closed or the cavity closes more rapidly because the air has ceased to enter is uncertain, but it has seemed to me that if the entrance

of air could be prevented the average case would be cured much sooner than it is. The small number of cases which I have had has prevented me from following up this idea as I would wish.

In my opinion the size and position of the opening have an important bearing upon the results of the operation. Elsberg showed that the size of the opening was an important factor in connection with the effects of experimental pneumothorax in dogs. If the opening made in most operations remained afterward as large as when made, I think that in many cases the lung never would expand, because it would be practically as large as that of the trachea or larynx, so that the external atmospheric pressure would remain constantly as great or greater than the internal pressure. It seems to me that the external opening must become smaller than the internal, or the tracheal diameter, before the lung can expand. After the usual operation the falling in of the soft tissues contracts the opening, which is further filled by the walls of the drainage-tube, and the lumen of the tube and the space around it are more or less choked up continually by the escaping discharge and fragments of fibrin. The air already in the empyemic cavity is becoming absorbed, and if a smaller quantity passes in than is being absorbed the external pressure on the lung is being diminished, so that the internal pressure coming through the trachea remaining the same the lung slowly expands. The excision of several ribs may be advisable to get nearer to the bottom of the cavity, but not to provide freer drainage. Through-and-through drainage by two openings with a tube in each and one higher than the other, in my opinion, is worse. The higher opening will always be admitting air freely, because the pus will be escaping entirely by the lower, so that the upper will be a strong factor in preventing reëxpansion of the lung. The term free drainage in connection with empyema has not the same meaning as with abscesses in other parts of the body. In many abscesses an incision is made from one end of the abscess to the other, and lays the cavity wide open. The Estlander operation for chronic empyema provides such an opening, and the result shows what is to be expected from it. The free admis-

sion of atmospheric pressure permanently prevents reëxpansion of a large portion of the lung, which causes a marked sinking in of the side of the chest and a corresponding loss of lung function. The small opening usually employed in acute empyemas, if placed at the bottom of the cavity, would provide drainage quite free enough for the average case.

Robinson says that in the early stages, with the drainage opening at the bottom of the cavity, suction drainage will not be necessary. I am, particularly, trying to show that this will be true for most cases. In 2 cases I made the opening by resecting a small portion of the tenth and in 3 cases the eleventh rib near the spine. By dividing the intercosal tissues in the latter to the top of the twelfth rib practically the very bottom of the cavity was reached. I did this because of observations made on the formalin specimen, which shows that in that case at least there would have been no difficulty in making such an opening, and I found in my cases that it was about as easy and safe as resecting the sixth or seventh rib. A small puncture wound was made in about the seventh interspace or where the aspirating needle demonstrated pus. A somewhat rigid probe or a long grooved director, curved at the end, was then introduced through this opening and the end made to seek the lowest limit of the empyemic cavity, which usually corresponded to the bottom of the normal pleural cavity near the spine. The curve permitted the end of the instrument to be turned toward the surface where by careful palpation it could be left by the finger outside over the corresponding interspace, usually the eleventh. A vertical incision was then made over this area, the eleventh rib exposed, and a small portion of it excised in the usual way. The probe or grooved director was then pushed outward through the pleura if it had not been already opened and a closed hemostatic forceps introduced at this point. The finger introduced through the opening thus made feels the twelfth rib below and the diaphragm at the bottom of the cavity. A drainage-tube is introduced after extending the opening by incision to the top of the rib below. A suture or two may be used to diminish the size of the opening

slightly, but free drainage must be the first consideration. During the operation I have placed the patient in the prone position advised by Elsberg, and have found it satisfactory. In one case, as I was about to make the preliminary incision for the introduction of the probe, I happened to look at a clock and I concluded to time the operation. When the drainage-tube was in and the gauze dressing applied about four minutes, not more than five, had elapsed. With such an opening the empyema will be perfectly drained in almost any position the patient is likely to occupy. The shoulders will probably be higher at all times than the opening, the advantage increasing the more nearly the patient assumes the sitting position. The opening is at the bottom of the narrow space between the diaphragm and chest wall, so that all of the discharge will constantly gravitate toward it and will tend to prevent the entrance of air in so far as it continues to fill the space in and around the tube. Usually I have not been greatly concerned about fragments of fibrin choking the tube, because I have felt that the drainage was free enough around the tube, and through it around any fibrin that became lodged in it. At any rate it will give no more trouble than with a higher opening. When we consider that we can in a short time evacuate the accumulation of weeks through the ordinary aspirating needle, it becomes evident that not much space is necessary with such a low opening for the continuous evacuation of the discharge as it forms. Nor have I been concerned about the falling in of the drainage-tube, since it is usually directed upward as well as inward, and would have to travel against gravity to get inside; and if it did so, would be kept by gravity at the opening so that it could easily be fished out with a forceps.

Agnew[1] applied the principle of dependent drainage in old empyemas with persistent sinuses, but maintained through-and-through drainage with a tube passed from one opening to the other. Ashhurst[2] employed the same method of drainage from the beginning, and says: "I have seen a number of cases in

[1] Agnew's Surgery, vol. i, pp. 344 and 345.

[2] Principles and Practice of Surgery, 1893, sixth edition, p. 410.

which this plan has been systematically carried out with complete success." It seems reasonable to assume that the lower opening drained all the pus in Ashhurst's cases, since pus could escape from the upper opening (the patient usually occupies the semi-recumbent position from the beginning and soon the upright) only if the cavity were full of pus, when the plan would hardly have been regarded as a complete success. The important point demonstrated by Ashhurst, in my opinion, is that an intercostal dependent opening only large enough apparently to pass the drainage-tube gave satisfactory drainage. But we have many surgeons who employ such an opening usually in the sixth or seventh interspace, especially in children. If the drainage is satisfactory through such an opening it ought to be better with the opening in the eleventh interspace. Only a scarcity of cases has prevented me from trying the low intercostal opening.

Some surgeons make efforts to remove fragments of fibrin at the time of operation. I have felt some concern about these fragments because they were in a pus cavity and were probably made up of necrotic material. On introducing my finger through the drainage opening in one case I found the pleural surfaces, parietal and diaphragmatic, covered uniformly as far as the finger could reach with a soft layer of fibrin about an inch in thickness, which could easily be swept off with the finger. It seemed obvious that the whole surface of the cavity was so covered and that it was all infected material. As it could not be removed I watched the after course of the case with much interest. The sinus closed and the patient was discharged in about five weeks. The layer of fibrin gave no trouble. It did not escape through the drainage opening unless it liquefied, which is unlikely. It is more probable that it remained and became organized into a thickened pleura. I did not discover such a deposit on the pleural surface in any other case. It suggests that in the pleural cavity we have the serous, scrofibrinous, and fibrinous varieties of inflammation, as in other serous cavities, and that the thickened pleuræ found in some old cases at operation may be due to the organization of such deposits.

Thomson and Miles[1] say: "Even in comparatively recent cases the pleura is thickened and covered with masses of flaky lymph, which may be an inch thick. In some cases of long standing this fibrinous exudate becomes organized and the pleura is greatly thickened."

In those troublesome cases in which the affected lung fails to expand after a prolonged period the last resort is a thoracoplasty, by the Estlander or Schede method, with or without removal of the thickened pleura covering the exposed part of the lung. Since the latter will not expand to obliterate the cavity the ribs overlying it are removed, permitting the weakened chest wall to fall in against the lung. The operation is frequently a severe one and the resulting deformity marked. The lung remains permanently collapsed in large measure, which with the sinking in of the chest wall produces a condition which is far from desirable. Robinson believes that these operations are too often employed in cases which are by no means beyond the possibility of reëxpansion. I believe that this is true. Ochsner[2] has made an important contribution to the treatment of these cases. In 14 cases he injected the cavity with Beck's bismuth paste, and all but 2, which were still under treatment, were cured, He has used it only in cases with sinues, although, he says, it has been suggested to aspirate the pus in the empyema and to inject the paste into the cavity through the cannula. While the method appears to be decidedly better than thoracoplasty, it would hardly be advisable to employ it to the exclusion of the usual drainage method, which results in a cure in most cases, with almost if not complete reëxpansion of the lung. Any paste retained permanently must be at the expense of the non-expansion of a corresponding portion of the lung. The cases calling for the Ochsner method, or thoracoplasty, are usually chronic not in the sense that they have long existed without being opened, but are cases which were opened in the acute or subacute stages and became chronic while waiting for the lung to expand. It is likely that in the chronic cases the lung has

[1] Manual of Surgery, fourth edition, ii, 318.
[2] Annals of Surgery, 1909, l, 151.

expanded considerably since drainage was begun, and therefore will require a much smaller quantity of the paste than if injected at the time the pus was first evacuated.

MASSIVE (PARIETAL) EMPYEMATA

Case I.—On February 13, 1906, I operated for the late Dr. Southwick on a girl, aged seven years, who had been rapidly accumulating a pleural effusion on the left side, soon after the crisis of a double pneumonia. The dulness was general and the dyspnea marked. Temperature, 101° F. Paracentesis was done in the seventh interspace in the scapular line for the relief of the dyspnea and for diagnosis. About a quart of purulent fluid was drawn. Two days later about 1½ inches of tenth rib was resected in the scapular line and more pus evacuated. The index finger introduced through the wound could not touch any part of the lung except a substance with a velvety feel in the direction of the spine. This was probably collapsed lung. When the finger was turned downward it could easily feel the bottom of the pleural cavity and the twelfth rib and came against the diaphragm. The opening was extended across the interspace to the top of the eleventh rib. Two large drainage tubes were introduced about five inches and sutured to the edges of the wound and the usual large dressing was applied. Three days later the tubes were removed and the finger again introduced. It could now touch lung which was evidently reëxpanding. As the opening was fairly large a portion of the lung could be seen. The patient was much improved. Temperature, 99° F. About one inch of each tube removed before replacing them. Eight days later and eleven days after the operation, when I saw her again, the finger recognized a decided gain in expansion of the lung. As on the last visit it passed into the fissure between the two lobes, which now had come closer together, and the external surface of the lung closer to the chest wall. One week later the finger failed to recognize any gain in expansion, and forced respiratory apparatus with two bottles was advised. In the following two weeks only slight gain was evident. On April 15, the tubes were removed, and two weeks later the sinus had closed.

Case II.—Man, aged thirty years. Right-sided croupous pneumonia began March 13, 1906, when Dr. Winslow Drummond was called. Had his crisis in nine days, which was followed by a short period of undisturbed convalescence, and this in turn by an increasing disturbance on the same side of the chest. Pleural effusion recognized March 26. A few days later aspiration tried at several points without

obtaining pus or other fluid. I saw the patient first on April 5 and by that time the dulness extended from the bottom of the chest upward to the third rib anteriorly and the spine of the scapula posteriorly. Breath sounds over the dull area were absent or indistinct. Patient was admitted to the University Hospital, in the service of Professor J. William White, April 6. On the same day a large veterinary hypodermic needle introduced in the ninth interspace, with an upward slant to avoid the diaphragm, and a few drops of thick pus obtained. On the following day about two inches of the tenth rib removed and empyema opened, a large quantity of thick pus escaping. A finger introduced felt a small portion of collapsed lung. Two large rubber drainage-tubes introduced about three inches and sutured to margins ot wound, a safety-pin being passed through each for further safety against falling in of the tubes. The space around and between the tubes was packed with gauze strips. Temperature dropped to normal in a few days and the patient's general condition rapidly improved. Air could be heard sucking through the opening until the end of the sixth week. The last tube was removed about the end of the eighth week, and two weeks later the sinus was closed. As it seemed that the large opening necessitated by the two tubes allowed too free an entrance of air I concluded to use only one tube after this.

Case III.—Boy, aged four years. Referred by Dr. B. F. Wentz. Admitted to the University Hospital, in the service of Professor J. William White, November 10, 1908. On the preceding day Dr. Wentz had found pus with the needle. The boy had been taken with left-sided croupous pneumonia about three weeks before, and had his crisis on the tenth day. He did not recover completely, and during the last few days was feeling much weaker than since the crisis had occurred. The dulness covered almost the whole left side of the chest, but there was slight resonance over the upper part of the scapula. On the day of admission, with the patient in the prone position, suggested by Elsburg, I excised about $\frac{3}{4}$ inch of the eleventh rib near the spine, in the manner already described. On introducing my finger into the cavity I detected an apparently uniform layer of soft fibrin, $\frac{3}{4}$ to 1 inch thick, covering the pleural surfaces, parietal and diaphragmatic, as far as the finger could reach. A small portion of it was wiped off with the finger and removed. No traces of this fibrin was found later in the dressings. An attempt was made at one time to employ negative tension drainage, with the method described by Schley, but a sudden rise of the temperature and illness of the patient caused me to give it up very soon. The patient was discharged from the hospital, with the tube removed, on December 6. Dr. Wentz reported the sinus closed December 14, thirty-four days after the operation

Case IV.—Boy, aged seven years, under the care of Dr. W. Drummond. Had severe erysipelas of the face and neck at nineteen months and a bilateral bronchopneumonia at five years of age. Since then has been strong and healthy. On February 12, 1909, was taken ill with influenza. Four days later had recovered sufficiently to return to school. On the following day, however, he was seized with a severe pain on the left side of the chest. On February 19, his temperature

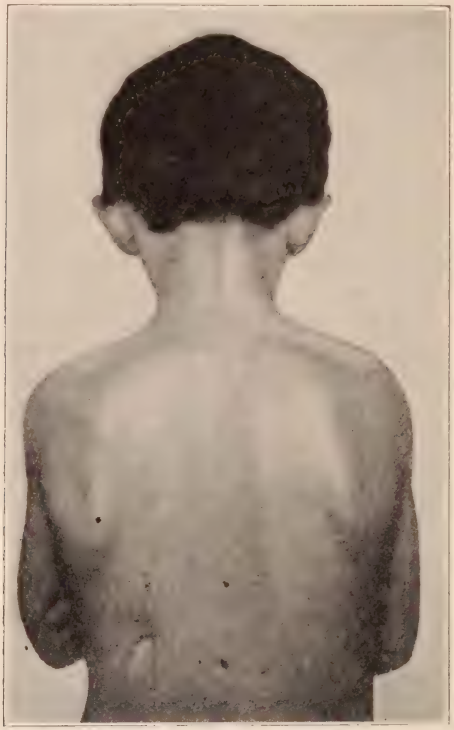


Fig. 2.—Scar at bottom of left side of thorax, posteriorly, after drainage of a massive empyema by excision of small portion of eleventh rib. Inferior angle of scapula normally is opposite seventh intercostal space.

was 101°; pulse, 98; respiration, 42; and croupous pneumonia was diagnosticated. Two days later the temperature had reached 105⅖° F., and the boy seemed septic, so that Dr. Drummond began to suspect an empyema and tried a small hypodermic needle without obtaining pus. On February 27, Dr. J. H. McKee was called in consultation, concurred in the diagnosis of empyema, and obtained a large quantity of pus by aspiration. On the following day, at the child's home, I excised about an inch of the eleventh rib near the spine (Fig. 2), with

the patient in the prone position. I happened to look at a clock as I was about to make the stab wound for the introduction of the exploratory probe and I concluded to time the operation. When the tube was in and the dressing was being applied from four to five minutes had elapsed. The temperature reached normal on the third day after operation. On the sixteenth day it rose suddenly to $102\frac{3}{5}°$ and the patient looked ill. The tube was removed and a plug of fibrin washed out when it was replaced. On the following day the temperature rose again, but after that there was no further trouble. The sinus was not closed until the ninth week, and in this case the sucking in of air on inspiration was heard until about two weeks before the sinus closed.

Case V.—Boy, aged eight years, under the care of Dr. C. L. Felt. Was taken with right-sided pneumonia October 17, 1910. Had his crisis in the usual time, but did not fully recover afterward. Dr. J. H. McKee was called in consultation and confirmed the diagnosis of empyema. On November 6, at the patient's home, I excised a small portion of the eleventh rib, as in the preceding cases. A large quantity of pus was evacuated and a half-inch rubber drainage-tube introduced and fixed to the margins of the wound. I saw the patient only a few times after the operation, Dr. Felt taking charge of the after-treatment. He reported the sinus closed January 3, 1911, about eight weeks after the operation

SMALL (INTERLOBAR?) EMPYEMAS

Case VI.—This case was reported in conjunction with Dr. J. H. McKee before the Philadelphia Pediatric Society in May, 1906. The patient was under the care of Dr. McKee, who made the diagnosis and located the pus collection. The following is the brief report of the case which I made[1] and the reference to the condition as a "localized or encysted" empyema was made before I had concluded that practically all acute empyemas are localized or encysted, "The opening in the chest-wall, made by a resection of about 1 inch of the sixth rib in the midaxilla on the left side, led directly into the cavity. That the empyema was a localized or encysted one was shown by the fact that the lung was adherent to the parietal pleura in the circumference of the empyema, which was demonstrated by the exploring finger. That the pus collection was in the fissure was evident from the depth of the cavity, which was approximately in the normal situation of the fissure dividing the two lobes of the left lung, and from the fact that

[1] International Clinics, 1906, cxi, 159.

the finger came directly upon the heart covered by the pericardium. Drainage was provided by two good-sized rubber tubes, introduced in the direction of the spine for about 5 inches. These were gradually shortened and healing was completed in five weeks. At the time of the operation the finger entered a roomy cavity, the walls of which were apparently under the influence of external atmospheric pressure. Soon afterward it could be determined through the wound by the eye and finger that the cavity was becoming filled by the expansion of the collapsed portion of the lung, this being aided by coughing and forced respiratory exercises."

Case VII.—Boy, aged two years, under the care of Dr. W. Drummond. Had apparently recovered from right-sided croupous pneumonia, which developed at the end of a three months' illness, including repeated "colds" and a glandular abscess of the neck. He did not remain well after the crisis, and on suspecting an empyema, Dr. Drummond called Dr. J. H. McKee in consultation on February 7, 1908. While dulness was found over the greater part of the left side of the chest it was most marked in the midaxillary region between the sixth and ninth ribs. Aspiration in the seventh interspace gave no pus, and it was repeated in the eighth interspace. No pus appeared on this trial until the needle was withdrawn, when immediately the inside of the bottle was spattered with pus spots, a small quantity appearing at the site of the puncture. On the following day, under chloroform administered by Dr. A. G. Tinney, and with the assistance of Dr. Drummond, I first made a stab wound with a narrow bistoury at the site of the aspiration puncture. A probe with the end bent slightly was introduced, but it could not be moved about freely inside the chest, and its end could not be turned downward. About an inch of the rib below was excised. The pleura was broken through by a grooved director, but only a small quantity of pus escaped. This opening was enlarged and a finger introduced, which found itself in a very small space, with the drainage opening at its lowest limit and the lung adherent to the chest wall all around the cavity. A drainage-tube with a half-inch lumen was introduced, sutured to the margins of the wound, and the dressing applied. The quantity of pus which escaped was small and about two tablespoonfuls of a fibrinous material escaped also. After the operation the child did not improve rapidly and continued to cough considerably, the temperature retaining the same septic type as before the operation. February 15 it was noted that he seemed to cough up a considerable quantity of fluid, which could not be inspected because it was immediately swallowed. It may have been the result of the breaking into a bronchus of a second pus collection, as he improved after it for a short time. February 24, the patient had been worse

for a few days, and an examination showed an area of dulness just anterior to the upper part of the axilla. Under ether anesthesia aspiration was tried here, but no pus was obtained. The finger was then introduced into the drainage opening, but found itself in the same circumscibed cavity as in the first operation, and brought out a considerable amount of the same fibrinous material as escaped at the first operation. On February 28, on account of the continued sepsis, the tube was removed and the cavity packed with iodoform gauze, but this was stopped and the tube reintroduced in a few days. The area of dulness above persisted, and on account of the continued symptoms of sepsis it was thought that there must be a second pus collection, indicating further operation. Dr. McKee was again called in, but advised against operation. The child was slowly improving, although the temperature still continued to be irregular and about as high as before the operation. Although the cavity was a small one from the beginning, on account of the septic character of the temperture it was thought advisable to allow the tube to remain. On April 24, it was concluded to remove the tube, regardless of the temperature. A few days later the temperature came down to normal and in about ten days the sinus was closed. The patient has continued well ever since and at the present time is in perfect health.

Case VIII.—Girl, aged eleven years, a private patient in the Germantown Hospital, under the care of Dr. Perry Cummings, who had called in consultation Dr. J. H. McKee. The patient had had right-sided pneumonia and the temperature had fallen by crisis, but had remained normal only a few days. When I first saw her, January 15, 1910, the relapse had lasted about a week, the temperature rising each afternoon to 104° and 105° F., and falling in the morning to about normal. The pulse remained at about 100 to 110 and the respiration at about 40. There was a localized area of dulness, reaching from about the third interspace above to the liver dulness below, and from the sternum to the anterior axillary line. About four days before a hollow needle had been introduced in this dull area, in the third and fourth interspace, without obtaining pus. It was thought that the needle had not passed into lung tissue. Air had rushed into the chest through the needle, indicating that the visceral and parietal pleuræ were not adherent to each other in the dull area. An *x*-ray taken seemed to show a small empyema nearer the posterior than the anterior surface of the lung. After a consultation it was concluded that a pus collection was present and that the lung should be exposed for further exploration and the evacuation of a pus collection if found. On the following day, January 16, the patient was given chloroform by Dr. McKee, and I exposed and removed subperiosteally

about 1½ inches of the fourth rib and cartilage. The pleura was then carefully opened after it was picked up by forceps. The lung was adherent to the parietal pleura by delicate adhesions. While separating these in the effort to locate the horizontal fissure of the lung the air began to enter the pleural cavity and the lung slowly to collapse. The lung was quickly grasped with a double tenaculum and drawn into the wound to plug it against the further entrance of air, and the lung was sutured to the circumference of the wound margins to permanently shut off the pleural cavity. A veterinary hypodermic needle was then introduced nearly its whole length into the lung four times in various directions, the piston being withdrawn each time. The results were unsatisfactory, only a small quantity of fluid being withdrawn each time, and that so mixed with blood that its significance could not be determined. The last puncture was made with a slight upward slant of the needle, and after removing it and expelling its contents into a basin it was observed that there were two small white spots in the fluid withdrawn. It was concluded that these showed the presence of pus. The actual cautery was then passed about 1½ inches into the lung in the direction of the last puncture, but no escape of pus followed it. A closed hemostatic forceps was then introduced into the lung opening, forced about an inch deeper, and the blades opened slightly. There immediately followed a gush of fluid, the nature of which could not be determined because of the blood present. The fluid flowed too easily to be pure pus, but it was not blood because the flow ceased immediately. None escaped into a bronchus. The quantity could not be determined, but according to my judgment there were about two ounces. The finger passed into the lung wound found what seemed to be a small cavity, with smooth walls which contrasted with the rougher surfaces of the rest of the wound. During the manipulations with the finger, which were gentle, air was heard entering the thoracic cavity and was evidently passing through the lung wound. It is likely that the collection of fluid was in a fissure and that the finger had broken down the circumscribing adhesions in it, so that the pleural cavity was again opened to the outside air through the lung wound. A strip of gauze was immediately packed into the lung wound, shutting off the new opening into the pleural cavity, and the whole wound was thus tightly packed to the skin surface. The skin wound was partly sutured, a dressing applied and held in place by a bandage. The patient was returned to bed in good condition. The external dressing was changed daily, but the packing in the lung wound was allowed to remain until the sixth day, when it was removed under nitrous oxide anesthesia. A rubber tube was then introduced and packed around with gauze, but the tube was removed on the following day and was not replaced

afterward, only light gauze packing being employed. Soon after this the packing was done away with entirely, the gauze being merely laid over the wound. The temperature fell to normal on the day after the operation and remained practically so during the convalescence, the wound being almost completely closed on February 14. The patient has since done very well and is now in good health

Case IX.—Girl, aged four years. Referred by Dr. B. F. Wentz. Right-sided pneumonia began March 27, 1910, and crisis occurred April 9. The convalescence progressed favorably four or five days. when the patient began to get worse, and there was detected some dulness and other signs of a pleural effusion, including fever and cough. May 18, the dulness reached as high as the third rib anteriorly. Aspiration was done in the seventh interspace in the midaxillary line, but only about two ounces of pus were obtained. The patient was admitted to the University Hospital May 19, in the service of Professor J. William White, and operation was performed on the same day. The exploratory probe introduced through the preliminary stab wound was too flexible to serve the purpose well but seemed to reach the eleventh interspace near the spine, although it was recognized that its bent end could not be felt under the eleventh interspace as well as in the preceding large empyemas. The eleventh rib was exposed and a small portion excised. The probe did not push easily through the pleura from the inside as usual, and the pleura was picked up with two forceps, as in opening the peritoneum, and was carefully incised. This exposed muscle fibers, which were evidently of the diaphragm. A grooved director was then substituted for the probe, and was pushed from within out through the lower opening by keeping it close to the chest wall. No pus appeared. The lower opening was then closed by sutures, a small drain being left in the lower angle. The ninth rib was then exposed and about 1 inch excised. The pleura was opened and a finger introduced, which found a small collection of pus, the escape of which was not accompanied by the usual inrush of air. Percussion showed that the dulness extended as high as before. A veterinary hypodermic needle introduced posteriorly in the seventh interspace withdrew pus. As this puncture was within the reach of the finger from the opening made by the excision of the ninth rib, the finger was introduced in the latter and by keeping it close to the chest wall it was passed upward toward the site of the last puncture when a freer escape of pus occurred than before and air was heard sucking into the chest on inspiration. The quantity of pus escaping, however, was not free enough to indicate the presence of the usual massive type of empyema. A drainage-tube about a half-inch in diameter was introduced and sutured to the margins of the wound, a safety-pin being passed through the tube for added safety. The tube

was removed June 6. The patient had left the hospital at the end of the third week with the tube in. The sinus closed during the last week in June, five or six weeks after the operation. The facts that the pus did not extend to the bottom of the pleural cavity and the quantity of pus present was only moderate in amount, together with the fact that it did not escape easily at first through the second opening, suggests the possibility or probability of an interlobar empyema in this case.

Summary. Our knowledge of the mechanical and pathological conditions existing in connection with empyemas has made little progress from observations made at operations or even autopsies. A formalin-hardened cadaver specimen of an empyema undisturbed by an opening during life is difficult to obtain, and therefore rare. For this reason such a specimen affords a particularly valuable opportunity for study. From a study of such a specimen and a small number of clinical cases the writer draws a few inferences which are at variance with the generally accepted views. They are as follows: We have not properly appreciated the extent and the nature of the adhesion formation developing in connection with empyemas, especially the acute variety. The massive parietal type extends usually to the bottom of the normal pleural cavity, and is not unencysted or general, but completely walled off above from the rest of the pleural cavity by adhesions. This explains the slight movability of the dulness on percussion, after changing the position of the patient, as well as the fact that the upper level of the dulness is not in a straight line as it should be if the fluid was unencysted and free to seek its own level. Skoda's resonance may not be due to relaxation of the lung above the pus, but to the fact that the functionating portion of the lung is doing compensatory work.

The probability is that adhesions between the lung and chest wall will not offer a serious obstacle to the reëxpansion of the lung, because they develop between parts normally in contact. The fact that a double empyema can be safely opened on both sides at the same operation is to be explained by the fact that the air admitted does not produce a total double pneumothorax, since it enters on each side only the firmly walled-off empyemic cavity.

Total collapse of the lung is prevented by the firm adhesions which protected the lung against the pus pressure before the drainage opening was made. There is no sudden or dangerous change of pressure on the thoracic organs from the usual rapid evacuation of the pus, but a gradual substitution of the pus by air, which has a pressure of fifteen pounds to the square inch.

The so-called encysted or localized empyemas are small, probably because they develop in the fissures of the lung or between the lung and diaphragm, and therefore because of the difficulty with which the pus is diffused in these situations.

The most important factor in preventing the obliteration of the empysmic cavity and closure of the sinus is the pressure of the air admitted through the drainage opening into the empyemic portion of the pleural cavity, where it neutralizes the expanding effect of the air coming through the trachea. Murphy overcomes this completely by aspirating the pus and injecting a formalin-glycerin solution, but his method has not yet received general approval. The drainage methods still prevail. The ideal drainage method is that based upon the suction or siphon principle. Its chief objection is that the devices in use for applying it generally leak air around the tube. We have not yet determined how rapidly an empyemic cavity, which is an abscess, may be permitted to close.

The size of the drainage opening has an important bearing upon the later expansion of the lung. The lung probably cannot expand until the entrance of air through the drainage opening is so diminished by contraction of the opening and blockage of the space in and around the tube by the escaping pus, that with absorption of the air already in the cavity there is developed a negative tension external to the lung sufficient to permit the normal internal pressure coming through the trachea to become greater than the external pressure. For this reason we cannot safely employ in empyemas the large drainage opening as in ordinary abscesses. The effect of the large opening in empyemas is shown after the Estlander operation by the permanent non-expansion of a considerable portion of the affected lung. An opening through the

eleventh rib or interspace of a given size will drain more perfectly than one at the usual level, and will better prevent the entrance of air, since the pus will be constantly escaping and tending to fill the space in and around the tube. There will be little or no danger from falling into the empyemic cavity of the drainage-tube, since it must travel against gravity to do so, and if this accident happened the tube probably could easily be reached and removed with a forceps, because gravity would prevent it from moving away from the opening. In some cases the much thickened pleura is the result probably of the organization of layers of fibrin deposited in the acute stage.

Of the five massive empyemas treated with dependent drainage it may be said that the time necessary for a cure was less in all than the average determined by Schädler, fourteen and one-half weeks, or the average in Fraley's cases, ninety days, and, therefore, there were no persistent sinuses. In the nine cases there were no deaths. The method deserves further trial and study.

DISCUSSION

Dr. John H. Jopson: I am sorry that Dr. Thomas did not describe more in detail his method of resecting. I assume that the section was made in the eleventh rib and well posteriorly. As we all know through-and-through drainage in empyema, as formerly employed by Dr. Ashhurst, is now almost universally abandoned for rib-resection, which is used even in very young children, and, of course, we all know the usual location—the seventh rib in the mid-axillary line or the eighth in the post-axillary line. This route was chosen only after a long series of observations and not arrived at by the study of a few cases. This location for resection is based upon well-founded anatomical and surgical reasons. Dr. Thomas tells us that in this postmortem specimen of massive empyema the costophrenic sinus was not obliterated. It has been my experience to find that this sinus is nearly always obliterated, at least in the axillary and post-axillary lines. If we resect below the seventh rib in the mid-axillary, and the eighth rib in the post-axillary line, we run the risk of injuring the diaphragm because this sinus may be filled

up by adhesions. If we resect farther back in the tenth and eleventh ribs, we may also encounter some adhesions between the diaphragm and the chest wall. The persistent sinus in these cases is not due so much to lack of drainage in this region as it is higher up in the pleura, and also to failure of expansion of the lung due to delayed operation.

Dr. Thomas, closing: I do not know how we are to determine before operation in a given case whether there are adhesions between the diaphragm and chest wall or not. All I know is that with the method which I employed it makes no difference whether such adhesions exist or not. What I have advised and done is to place the drainage opening at the bottom of the empyemic cavity, wherever that may be. In the five large empyemas in which I applied the method, the empyemas were found to extend to the bottom of the normal pleural cavity in every one, and in the small empyemas the opening was placed at the bottom also. The method of determining this level was essentially the same as that employed by Prof. Ashhurst. He inserted a long probe with a curve in the end introduced, by means of which he could press the tip inside against the chest wall and feel it through the wall at the bottom of the empyema. After making an intercostal opening at this level, he tied a rubber drainage tube to the eyed-end of the probe above, and by means of the probe drew the tube through from one opening to the other, leaving the tube projecting at both openings. I have used the probe in the same way except that I did not draw it through. The tube was introduced only through the lower opening, the upper small one being allowed to close immediately. I am firmly of the opinion that in the majority of the large empyemas, which might be called the parietal in contrast to the interlobal type, the fluid falls immediately to the bottom of the normal pleural cavity, as Donaldson said, and prevents the development of adhesions in the costophrenic sinus, except, perhaps, where a small portion of the lung became adherent before it was lifted by the accumulating pus, as occurred in my specimen. It is quite another thing to say that the costophrenic sinus becomes obliterated by adhesions after the operation. I believe that this can happen after operation up to a certain point, but that it will not interfere with effective drainage through an opening at the bottom of the cavity made at the time of the operation and kept open afterward by the tube.

REVIEW OF 534 OPERATIONS UPON THE MAMMARY GLAND[1]

By JOHN B. DEAVER, M.D., Sc.D., LL.D.

There are two important epochs in the surgical history of mammary carcinoma. The modern operation, based on the knowledge of the lymph-vascular drainage of the breast has proved of value from the standpoint of the ultimate cure, in the proportion of the introduction of antiseptics and asepsis to its primary mortality. This is the more remarkable in view of the extensive operation now performed, so that the reduction of the primary mortality of 25 per cent. in pre-antiseptic days to less than 1 per cent. with modern technique, is quite in proportion with the reduction in local recurrences from 65.6 per cent. to 6.5 per cent. Elaboration of Halsted's principles has carried the operative incision farther and farther afield of the primary focus of disease, until the limitations of surgery must surely have been reached, and with results that, uncompared with results of past decades, are highly disappointing. Why must the ultimate cancer be sought for in remote tissues of cervical, thoracic, and abdominal areas, and usually in vain, when it is the concensus of medical opinion, that cancer is primarily a local disease, often, perhaps always, preceded by a pre-cancerous state of the tissues, and invariably amenable as such to surgical treatment? To whom can we turn for explanation of the inoperability of 25 per cent. of cases presenting themselves to the Mayo clinic, of the inoperability of 32 per cent. of cases of superficial malignancy seen by the surgeons of Pennsylvania in 1911, or of the presence of extensive ulceration

[1] Read December 4, 1912.

and metastasis in 31 of our last 200 cases? Is it the memory of an ancient teaching that prompts physicians to advise the patients to accept operative treatment as a *dernier ressort* in mammary carcinoma, when this lapse of time has removed its only chance of cure? Or has surgical practice in accepting advanced cases for operation cast an opprobrium on its methods, established a widespread belief in their futility and deepened the conviction that operation is only to be accepted when this very skepticism has rendered it useless. Is it the ignorance of the laity in regard to the serious nature of lumps, whether painful or not? Or is it the apathy of the practitioner, or over confidence in his ability to differentiate the benign from the malignant in its early undifferentiable stages? No doubt a number of factors are potent in the production of this evil, while the fact remains that surgeons see large numbers of inoperable cases of cancer of both superficial and deep origin, the great majority of which could have been cured if diagnosticated early and treated properly.

Five hundred and thirty-four patients have been operated on in the German Hospital Clinic for tumor of the breast, since 1898, as follows:

Carcinoma	370
Fibro-epithelial	123
Chronic mastitis	24
Sarcoma	11
Tuberculosis	6
	534

An analysis of the last 200 cases of carcinoma and 100 cases of fibro-epithelial tumors, of which the records are complete, with the end results of all cases which we are able to trace, forms the material from which the statistics to follow are computed.

INCIDENCE OF CANCER AND THE PRE-CANCEROUS TISSUE STATE

The statistics of all countries show a steady increase in the incidence of cancer. In New Zealand, which country has the

lowest death rate from cancer, there is a steady increase, proportionate as shown by Hislop and Fenwick with the increase of other countries. The death rate in the United States had risen from 47.9 per 100,000 in 1890, to 73 per 100,000 in 1907, almost the same percentage average increase during this same period for eight European countries. The international classification of the cause of death attributes 8.5 of the deaths from cancer to mammary involvement. Estimated from this, 5882 of the persons dying from cancer in the United States during the year 1907 had primary involvement of the breast. Coincident with this increase in the incidence of cancer, there has been a steady increase in our knowledge of the predisposing states of the tissues to malignant degeneration, and in a measure of their underlying cause. Keen's demonstration, twenty years ago, of the degeneration of pigmented moles into carcinoma, foreshadowed the general recognition of certain connective tissue changes predisposing to malignancy. Victor Bonney believes that cancer cannot originate from epithelium lying on healthy connective tissue. He states that destruction of the elastic tissue fibers of the basement membrane is the initial step toward malignancy, a result produced in the majority of cases of cancer of the breast by chronic cystic mastitis, especially of the proliferative type. As causative of this type of mastitis, Handley mentions,

"(*a*) Infection *via* the ducts.

"(*b*) Toxic products of metabolism.

"(*c*) A morbid deviation in the physiological process of involution and evolution which normally occurs with the sexual crises, puberty, menstruation, lactation, pregnancy, and the menopause."

Beadles found this condition in the non-cancerous portions of a hundred specimens of carcinomatous breast, and shares the opinion of E. T. Paul and Victor Bonney that it is in constant association with early mammary carcinoma. Its absence in the late stages they ascribe to overgrowth of the cancer elements. From clinical evidence, Handley estimates the percentage of antecedent mastitis as 30 per cent., comparable with the 39 per cent. of superficial cancers, in the report of the Pennsylvania

Cancer Commission, giving a history of chronic irritative conditions antedating the development of malignancy. MacCarty, Speese, Bloodgood, Judd and others associate chronic mastitis with carcinoma in from 25 to 75 per cent. of cases.

Benign tumors are said to constitute 11 per cent. of all breast tumors, of which 7 per cent. are of the fibro-epithelial type.

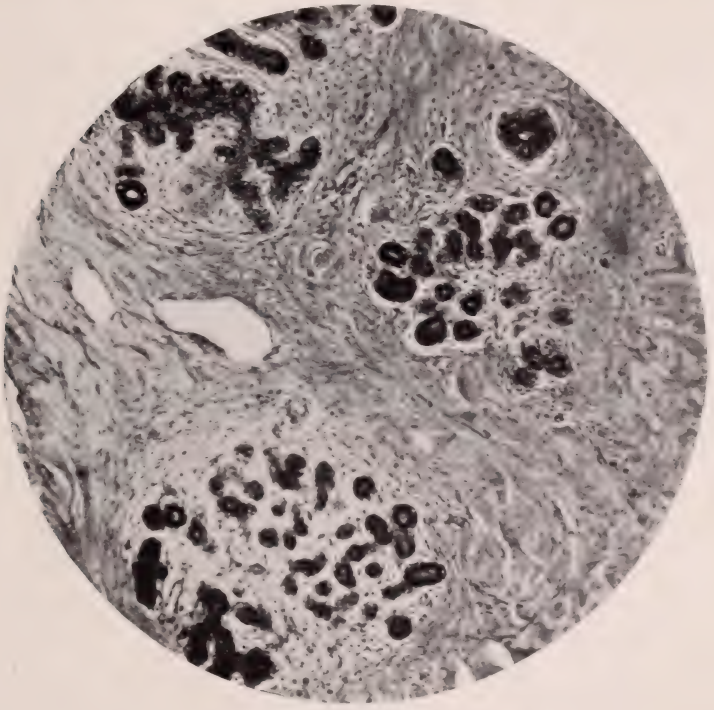


FIG. 1.—Fibro-adenoma of the breast.

This estimation is perhaps true when based on hospital statistics, but benignancy is in much greater proportions in early stages. According to Judd 30 to 35 per cent. of benign tumors of the breast suffer malignant degeneration. In one of our cases a small tumor was excised from the left breast and diagnosticated "fibro-adenoma" macroscopically. Fig. 1 shows the appearance of the primary growth. The patient returned in six months with

extensive carcinoma. Fig. 2 shows carcinoma in the scar from the original operation. This case while it illustrates a clinical and pathological mistake serves to emphasize the important fact that a growth may appear clinically and pathologically benign when already in a small area malignant transformation has taken place.

Fig. 2.—Recurrent carcinoma in scar.

The influence of trauma, whether of transient nature or long-continued irritation, is much disputed. Of 115 authors writing in the years 1901 to 1909 inclusive, thirty did not assign trauma as a cause of cancer. Phelps states that only 9 per cent. of authors since the middle of the last century believed that transient injury might be the cause of malignant change. Thirteen per cent. of our cases gave a definite history of injury, usually transient in nature.

In one instance bleeding from the nipple, said to be of traumatic

origin, recurred at irregular intervals for one year before the clinical evidence of malignancy appeared. Williams states that 25 per cent. of 137 cases gave a history of trauma, and that in 1 in 1000 cases the cancer immediately followed its receipt. With increased knowledge of pre-cancerous tissue states, the ascribed influence of bacterial and traumatic inflammation has a more rational basis. Nor can the frequent occurrence of the disease in the absence of such influence render it equivocal, for in the light of present knowledge, we must consider trauma, with other causes of inflamamtion, as a factor predisposing to cancer through the destruction of the normal fibro-epithelial relationship.

Symptoms and Diagnosis. Thirty-seven of the relatives of our malignant cases, and 22 of those of the benign cases died of cancer, in 13 per cent. of each type affecting the breast. Various authors state that from 5 to 30 per cent. of operated cases of cancer give a family history of the disease, usually affecting the breast. The occurrence of mammary carcinoma in four or more members of a family, a history given by two of our cases, would indicate hereditary influence, but this must be considered a developmental predisposition.

As regards sex, the number of males varies from 6 per cent. (Rodman) to 2.82 per cent. (Billroth): 1.5 per cent. of the malignant and 2 per cent. of the benign cases of this series occurred in males. This low percentage is in keeping with the general rule that malignancy rarely affects vestigial structures, of which nature the male breast is to be considered. It also lends weight to the influence of changes incident to function in the female breast in the development of precancerous tissue states. Fibro-epithelial tumors occur at all ages, while no authentic cases of cancer have been observed before puberty. The cases so reported have been, as Billroth believed, the case of Birkett in an eight year old girl to have been sarcomatous in nature. Carmichael and Everhard Home reported bilateral carcinoma in patients twelve and fifteen years of age. The age incidence in our cases is shown in the following table:

Cancer cases. Average age . . . . . 49.6 years
Fibro-epithelioma. Average age . . . . . 34.5 years

Cancer			*Fibro-epithelioma tumors*		
		Per cent.			Per cent.
20 to 30	5	2.5	10 to 20	6	6
30 to 40	32	16.0	20 to 30	25	25
40 to 50	65	32.5	30 to 40	32	32
50 to 60	65	32.5	40 to 50	25	25
60 to 70	19	9.5	50 to 60	6	6
70 to 80	14	7.0			
	200	100.0		100	100
		Years.			Years.
Youngest female		22	Youngest female		14
Youngest male		25	Youngest male		19
Oldest female		76	Oldest female		61
Oldest male		78	Oldest male		23

Menopause

		Per cent.			Per cent.
Before	95	47.5	Before	91	91
After	73	36.5	After	7	7
(Males	3	1.5)	(Males	2	2)
No record	29	14.5			
	200	100.0		100	100

Marital

		Per cent.			Per cent.
Single	34	17.0	Single	49	49
Married	163	81.5	Married	49	49
Males	3	1.5	Males	2	2
	200	100.0		100	100

Parity

	Per cent.		Per cent.
110	55 parous	24	24 parous
90	45 nulliparous	76	76 nulliparous
200	100	100	100

Diagnosis. When a positive diagnosis of mammary carcinoma can be made, the hope of operative cure is often in vain, for the classical signs are usually unmistakable evidence of extensive metastasis. Mistakes, however, in the clinical diagnosis in advanced cases are constantly made and, as Hort has shown, especially in young subjects. In a series of 300 cases occurring before the twenty-fifth year of life, this author states that 128 were not

diagnosticated clinically, and further, that 582 of 3532 cases at all ages were sent to the London hospitals with wrong diagnoses. In the early malignant changes of primarily benign breast tumors, and in carcinoma arising *de novo*, it is as impossible to diagnosticate the condition as in the early analogous changes in the base of a chronic gastric ulcer. The reproach to him who fails to recognize obvious cancer is no greater than that of the physician who waits for obvious signs. A small minority of cases progress insidiously to extensive dissemination in the absence of symptoms. One of our cases was sent to the hospital on account of severe upper abdominal pain. Examination showed extensive involvement of the liver, metastatic from a small nodule in the breast (right) of which the patient had been entirely unaware. Two patients first noticed nodules in the axilla and had never suspected any trouble with the breast. In the great majority of cases, the disease runs a symptomless course in its early stages, and the first suggestion of trouble comes with the accidental discovery of a "lump" in the breast. It is our opinion that if, with the discovery, the patients were subjected to immediate radical operation a much larger percentage of cures could be obtained. The initial symptoms in this series were as follows:

Cancer		Per cent.	*Fibro-epithelioma tumors*		Per cent.
Lump	156	78.0	Lump	85	86
Pain	18	9.0	Multiple, 5		
Eczema of nipple	3	1.5	Bilateral, 3		
Bleeding from nipple	2	1.0	Pain	10	10
Ulcer of nipple	3	1.5	Spontaneous, 7		
Soreness of nipple	3	1.5	At menstrual periods, 1		
Retraction of nipple	2	1.0	After trauma, 2		
Serous discharge from nipple	1	0.5	Aching	1	1
Increase in size of breast	2	1.0	Burning	1	1
Lump in axilla	2	1.0	Tender at the menstrual periods	2	2
Stinging sensation	2	1.0			
Tender axilla	1	0.5			
Dimpling of skin	2	1.0			
Sinus	1	0.5			
Pigmented mole	1	0.5			
Pain in upper right abdomen	1	0.5			
	200	100.0		100	100

These statistics refute the assertion of Billroth that the initial symptom of mammary cancer is "lancinating pain which sometimes precedes the palpable induration by months." The disease in one of the cases in this series continued to extensive ulceration in the entire absence of pain. This symptom is common, however, in the late stages. Sixty-five per cent. of the malignant cases and 25 per cent. of the fibro-epithelial type suffered pain from several months to several years after the discovery of the tumor. The pain has been described as slight, moderately severe, or excessive, of a sticking, drawing, aching, and burning character, knife-like and shooting into the arm, axilla, neck, abdomen, or chest, and in several instances was complained of as affecting only the axilla and abdomen. Trauma, infectious diseases, the menstrual periods, and damp weather were given as influential in its production. As a symptom in the late stages, pain may be general affecting especially the extremities and due either to toxemia, widespread metastasis, or both. In early malignancy of the breast pain must be considered a rare symptom. Occurring late in the disease, it is the language of reproach to the patient for concealing her malady, to the physician for ill advice, or to the surgeon for operative procrastination. Translated it often means "too late."

Physical Signs. The opportune time for surgery in mammary carcinoma is in the presence of a symptomless, freely movable mass. That patients are rarely seen in this stage of the disease is indicated by the following tabulation of physical signs in our cases:

Physical Signs, Side

Cancer		*Fibro-epithelioma tumor*	
Right	94	Right	51
Left	95	Left	46
Bilateral	11	Bilateral	3
	200		100

11 of the benign cases had previous operations on the other breast, in 4 instances for carcinoma.

Mobility (on chest wall)

Movable	157	Movable	99
Fixed	43	Fixed	1
	200		100

Skin Involvement

Skin involved	141	Skin involved	3
Skin not involved	59	Skin not involved	97
	200		100

Retraction of the Nipple

Retraction	107	Retraction, 1 congenital	5
Not retracted	93	No retraction	95
	200		100

Discharge from Nipple

Discharge	6	Discharge	1
No discharge	194	No discharge	99
	200		100

Ulceration

Ulceration	31	Ulceration	1
No ulceration	169	No ulceration	99
	200		100

One showing ulceration, bilateral. Both sides involved.

Involvement of the Axillary Lymph Nodes

Palpable	74	Palpable	9
Non-palpable	126	Non-palpable	91
	200		100

13 of the 74 palpable enlargements non-cancerous.

The 9 palpable cases proved non-cancerous microscopically.

In no case of this series were the lymph nodes of the opposite axilla of the supraclavicular spaces palpably enlarged. Without considering general symptoms at length, we would draw attention to the frequent occurrence to a gain in weight of women with cancer of the breats, incidential to metabolic changes with the menopause.

Location. Fibro-epithelial tumors are said to involve the sternal half of the breast most frequently. In this series, as indicated by Figs. 3 and 4, both malignant and benign types of tumor were most frequently found in the upper and outer portions of the breast.

FIG. 3

Axillary half 54%

Central 11%

Upper half 45%

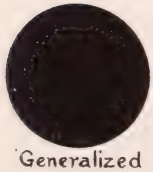
Upper inner 4%

Sternal half 5%

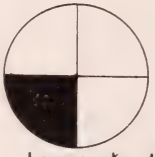
Upper outer 28%

Lower half 29%

Lower inner 0%



Generalized 3%



Lower outer 17%

FIG. 4

Axillary half 38.7%

Central 19.7%

Upper half 38.7%

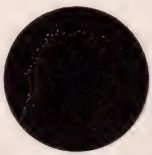
Upper inner 5%

Sternal half 10.2%

Upper outer 29.2%

Lower half 12.2%

Lower inner 2.7%



Generalized 9.2%

Lower outer 9.2%

Metastasis. Palpably enlarged axillary or supraclavicular lymph nodes in the presence of a mammary growth does not necessarily mean carcinomatous involvement. In both chronic cystic mastitis and cancer these nodes undergo certain changes affecting especially their germinal centres, and leading the hypertrophy, which has been styled the pre-cancerous adenopathy. Palpable axillary nodes were noted in 9 of our benign cases and proved free of malignant change by microscopic examination. Finsterer found the same condition 7 times in 64 cases of benign neoplasms.

The microscope proved the absence of metastasis in 6.5 per cent. of the 37 per cent. of malignant cases in which axillary enlargement was found by palpation, although in 62 per cent. of the cases, metastasis to these nodes was found microscopically. Bull stated that 65 per cent. of cases when seen by the surgeon have palpable axillary lymph nodes. Halsted reported two cases of mammary carcinoma in which the axillary enlargement in one instance preceded the development of, and in the other progressed in the entire absence of any demonstrable mammary neoplasm. The two similar cases in this series had definite growths in the breast when the axillary masses were discovered, although the patients were unaware of their presence.

Looking with Halsted on metastasis as "the centrifugal spreading of a growth in direct continuity with the primary focus," the axillary enlargement in the latter type of Halsted's cases must be proved separate from a small primary focus in the axillary extension of breast tissue, called by Stiles "the axillary tail."

In a number of cases the neoplasm involved the upper inner quadrant of the breast; and we believe the entire freedom of lower cervical lymphatic involvement in this series would not have been noted if the lymph channel, described by Poirier and Cuneo, were effective in draining this area. Nor does the freedom of attachment of the tumor to the pectoral fascia indicate non-involvement, for not only is this fascia involved before adhesion occurs, as Heidenhain showed in 1889, but both muscles and the intermuscular lymphatics as well, though this is more rare. The

same is no doubt true of the digitations of the serratus magnus muscle arising from the fifth and sixth ribs, in involvement of the lower outer quadrant, which overlies them. Henderson failed to show this relationship, and there are no other studies available to indicate such involvement, yet Handley agrees that in cancer of the lower axillary quadrant, these digitations should be removed.

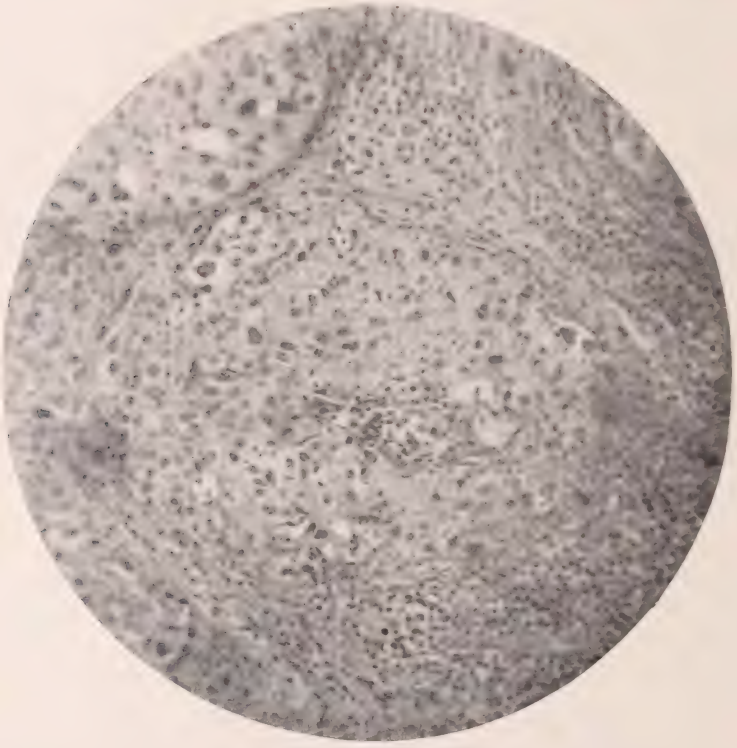


FIG. 5.—Metastasis of carcinoma of breast to region of angle of scapula.

We removed a mestastatic nodule from the tissues over the inferior angle of the scapula on the diseased side (Fig. 5). It was probably metastatic along lymphatics in the fascia covering the latissimus dorsi muscle, from the primary focus in the upper outer quadrant of the breast. Liver involvement, the frequent occurrence of cancer deposits in the humerus and thoracic cavity, are the result

of lymphatic permeation. Blood-vascular embolic infection, the possibility of which is sometimes denied, probably explains distant metastases. Metastatic carcinoma to the breast is exceedingly rare, except from its fellow. The same is true of primary bilateral mammary carcinoma.

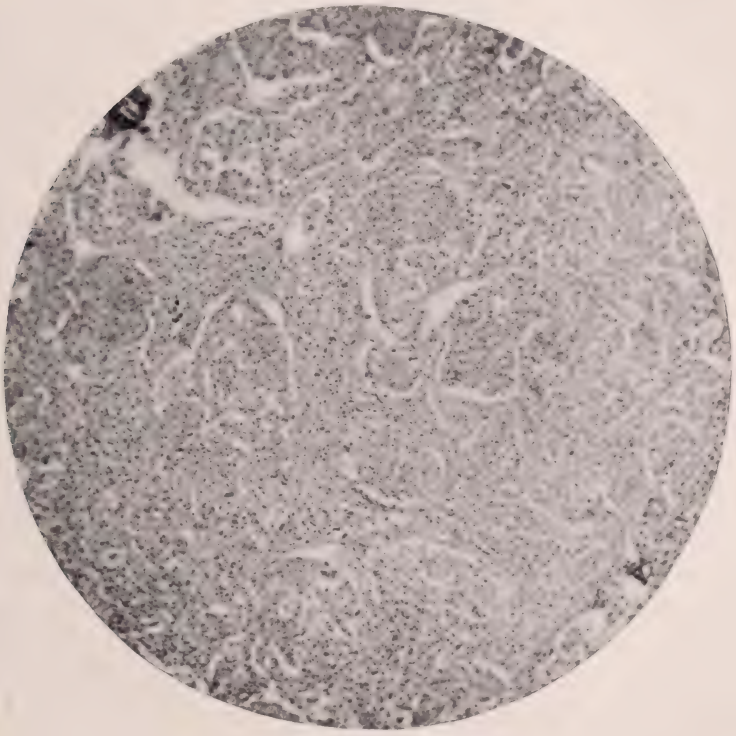


Fig. 6.—Adenocarcinoma of jejunum

Late Recurrences. We have noted apparent recurrences twenty years after the removal of a cancerous breast. In one case of this series the left breast was removed six years before signs of involvement of the right breast appeared. In another carcinoma appeared at the site of a vaccination scar three years after amputation of the breast. We resected a malignant jejunum and associated involved lymph nodes (Fig. 6) from one of the

cases of this series three months before the discovery of a breast tumor. The patient was admitted to the German Hospital in February, 1907, with symptoms of pyloric obstruction, from which she had suffered since September, 1906. At operation an annular carcinoma was found, involving two and a half inches of the jejunum, and situated eight inches distal from the duodeno-jejunal flexure. The mesenteric lymph nodes draining this area were palpably enlarged. The diseased portion of the jejunum was resected and an end-to-end anastomosis made, but insufficient blood supply necessitated resection of the primary anastomosis. A lateral anastomosis with posterior gastro-jejunostomy to its distal side was then performed with satisfactory results. The enlarged lymph nodes in the immediate area of the resection were removed. The patient recovered promptly, but returned to the clinic in April, 1907, complaining of sharp pain around the nipple of the left breast. This pain came on suddenly five weeks before admission and was associated with a rapidly growing lump in the upper outer quadrant of the breast. A simple amputation of the breast was performed. The specimen proved to be an adeno-carcinoma. The patient writes us (August 15, 1912) that she enjoys perfect health.

From another patient we removed a carcinomatous uterus seven years before the radical operation for similar trouble with her breast, but of this case we have no word. Matas has reported local recurrence twenty-four years after excision of the breast Of 35 cases of late recurrence, Rotter found 6 in the other breast. Most of the so-called late recurrences are new processes without relationship with the previous trouble.

Röntgen irradiation has no place in the treatment of operable carcinoma of the breast. Pre-operative application of the rays to remove outlying metastases before operative removal of the primary focus is as useless as local treatment of vesical tuberculosis in the presence of primary renal involvement. As a prophylactic measure against post-operative recurrence, or in cases of local recurrence which on account of its position and extent or the debilitated state of the patient precludes further operative

interference, and in inoperable cases of cancer of the breast, the *x*-ray is the only therapeutic measure of value. Bogg's report of five apparent cures in twenty inoperable cases, with improvement in the remaining ones lends encouragement to patients with cancer involvement beyond surgical relief; to state the value of Röntgen irradiation in these conditions, in terms of finality, is impossible. Dr. G. E. Pfahler, and other eminent Röntgenologists, recommend the treatment under the circumstances above named, and for this reason, patients in the immediate post-operative period, with local recurrence impossible of removal, or with inoperable mammary carcinoma should have the benefit of their wide experience.

Prognosis. In the 493 cases of carcinoma and fibro-epithelial tumors operated in the German Hospital since 1898, there were three deaths. Endocarditis developed after excision of a small "fibro-adenoma," the others died of uremia and pneumonia following the radical operation for carcinoma.

The average primary mortality is approximately 1 per cent. with the radical operation. As concerns the possibility of cure in cancer of the breast, the prognosis depends on those factors, including length of time the disease has existed, the age of the patient, type of tumor, etc., which contribute to dissemination of the disease, and from the technical standpoint the ability to remove without cancer implantation, the glandular, lymphatic, fascial, and muscular tissues involved in the malignant process. The average length of time the disease has existed in this series was thirty months. In the cases giving a family history of cancer 22.9 months had elapsed between the initial symptom and surgical consultation. In the cases well three or more years after radical treatment sixteen months had elapsed on the average before operation. Freedom from recurrence was obtained equally in cases of long and those of short duration at the Massachusetts General Hospital, and Greenough's tables indicate that bilateral involvement involved axillary and lower cervical nodes, ulceration and adhesion of the tumor to the skin and chest wall with the medullary type of tumor, and in a young subject offers the

most unfavorable prognosis. Of our cases well after three or more years only 12 per cent. had retraction of the nipple, and 18 per cent. attachment of the tumor to the skin, but in no instance was the tumor attached to the pectoral fascia. We produce the tables from Greenough's studies.

Total cases		376
Well after three years	64	
Died of other diseases after three years	7	
	71	18.9 per cent. successful

Primary mortality of 416 cases 3.6 per cent.

		Per cent.
Tumor adherent to the skin	262	16.0 successful
Skin not involved	71	32.0 successful
Tumor adherent to chest wall	45	11.0 successful
Movable on chest wall	194	21.0 successful
Axillary glands palpable	236	12.0 successful
Axillary glands not palpable	117	19.0 successful
Bilateral involvement	6	all fatal
Ulceration present	60	6.5 successful
Non-ulcerative	316	21.0 successful
Medullary cancer		16.0 successful
Scirrhus		23.0 successful
Adenocarcinoma		47.5 successful

In 40 cases with enlarged supraclavicular glands only two survived, and in these the glands free of cancer (microscopically) 52 per cent. remained free of local recurrence. Forty-one of 290 cases of recurrent carcinoma lived over three years after operation, and 13 per cent. of these never showed local recurrence. In 1907 Halsted reported 42 per cent. of 210 cases cured after the three year limit.

In Halsted's early cases two in three were cured with the radical operation, which in those cases with metastases to the axillary nodes, the late cases, three in four died of early recurrence. He reports 7 per cent. of cures in 44 operations with dissection of the supra-clavicular space: 90.9 per cent. of the 59 cases who died of early recurrence in our series had palpable axillary involvement. Of 16 living after the three-year limit, 25 per cent. had palpable lymph nodes in the axilla. The end-results in 119 of

our cases are as follows: Of the fibro-epithelial tumor cases, 44 have been traced and of these 41 have remained well for an average period of six years. Two have had operations for similar tumors in the opposite breast, and one case diagnosticated fibro-adenoma both clinically and microscopically had early malignant degeneration of the breast (see Figs. 1 and 2) and died of recurrence after radical operation. Sixteen, or 21.3 per cent. of 75 cases of carcinoma have passed the three year limit and are free of recurrence on an average of seven years. Thirty-seven died of recurrence, and four of causes other than cancer. Six others have recurrence at the present time; while the remaining ones are apparently well, but sufficient time has not elapsed to make this certain. These statistics, though meagre, indicate in a general way the present status of mammary carcinoma as regards the prognosis of operative treatment. Approximately one in five is permanently relieved of the disease by the radical operation, though it must be confessed that the inability to secure reliable data makes even this percentage questionable. This percentage would probably be materially lessened if the cases pronounced inoperable, and whose names do not appear on hospital records, were included in the statistics. As mentioned above, these results are highly disappointing unless compared with the results of past decades. When popular opinion demands immediate operation upon the discovery of a lump in the breast; when physicians are taught to think of breast tumors in terms of operability; and when misguided humanitarianism no longer prompts the surgeon to attempt injudicious operations, the present lack of faith in the surgery of this disease will give way to a healthy optimism.

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DISCUSSION

Dr. James Tyson: I should like to ask Dr. Deaver whether the percentage of carcinoma is increasing. We all know that there are many more cases of cancer than there were fifty years ago, but I have had some difficulty in getting a definite answer to the question, "Is the ratio of cases increasing?"

Dr. Astley P. C. Ashhurst: I am glad Dr. Deaver approves of the *x*-ray treatment after operation, and I was glad to hear what he said of this treatment before operation. Personally, I believe its employment after operation is of much value. I have in mind now a woman who came to the Episcopal Hospital over three years ago with what was considered "inoperable" carcinoma in both breasts. Upon Dr. Frazier's advice I amputated both breasts, merely to relieve her of the sloughing area, and without exposing the axillæ, in both of which lymphatic masses were palpable. The *x*-ray treatment was then given for two or three months, and not until this was discontinued did she get a slight recurrence in the cicatrix on the left side. This was excised by Dr. Neilson in January, 1910. The *x*-ray has been used regularly since then by Dr. Thos. S. Stewart, and there has been no local recurrence. There is now metastasis in the soft parts of the right thigh, but this is not ulcerated; she has no pain, and under treatment with the *x*-ray this mass is becoming smaller. Thus she has been kept comfortable for more than three years, and is in good general health for a woman aged seventy years.

Dr. John B. Roberts: I should like to ask Dr. Deaver what he would do in the case of the rather young woman, aged between twenty and thirty years, when small nodules appear in the breast, whether he excises a small section for microscopic examination before doing the radical operation. These cases are the ones that trouble some of us a good deal in determining whether we shall mutilate the young woman before making a microscopical diagnosis of a small hard mass. My own feeling is that it is better to take out the lump with as little mutilation as possible, have it submitted to microscopic examination, and then, if necessary,

go ahead with the radical operation. I rather gathered from his paper that he would do a pretty radical operation at the very first in most cases. Some of us would also in women in the neighborhood of the menopause.

Dr. George Erety Shoemaker: I was about to ask the same question asked by Dr. Roberts. I would ask further whether Dr. Deaver fears the dissemination or inoculation effect of excising portions of a growth still connected with the lymph and blood channels of the patient, in order to make frozen section diagnosis during operation?

Dr. Deaver, closing: I can say in answer to Dr. Tyson's question, unmistakably, yes. I believe that carcinoma is decidedly more common now than formerly, and I believe that statistics being prepared from year to year and from years to years bear out the truth of the statement. I am glad that Dr. Ashhurst agrees with me in regard to the value of *x*-ray after operation rather than before. I am fully in accord with him. I think its employment before operation is a great mistake, but regret to say that the practice is not very uncommon. I would limit the *x*-ray to the postoperative treatment. I have questioned whether it had anything to do with intestinal metastasis. While I do not believe that it has, I have seen a few cases which, after vigorous *x*-ray treatment, have shown metastasis to the liver and stomach, and this naturally has brought up the question.

In answer to Dr. Roberts I would say that we make it a rule in the German Hospital clinic in all tumors, not only of the breast, but all tumors, to have a frozen section and a microscopic diagnosis made then and there. We adhere strictly to this rule, not only in breast tumors, but tumors of other portions of the body. Under this order I would not take a breast off unless sure of my diagnosis. If there is any question concerning diagnosis the examination of the frozen section is followed by an examination of the stained section. This also answers Dr. Shoemaker. In regard to the relative value of the frozen and the stained section, I have as much faith in the former as in the latter. These examinations are all made by our house doctors, and in only one or two cases did Dr. Pfeiffer have occasion to change the diagnosis. Those familiar with the work done at Rochester know that the frozen section is regarded as more reliable than the stained. I feel that all clinics should have in operation the system of the frozen sections for diagnosis.

RESULTS WITH TUBERCULIN (DIXON'S) IN ADVANCED CHRONIC AMBULATORY TUBERCULOSIS[1]

By A. P. FRANCINE, A.M., M.D., and H. J. HARTZ, M.D.

It is unnecessary in connection with the following report to discuss even by way of introduction the broad question of the use of tuberculin in any of its many relations, as for instance its availability in dispensary practice, which is, we believe, fully established. Our purpose is to report briefly the results in a series of ambulatory cases, principally because in these cases there was a peculiarly favorable opportunity to eliminate other causes of improvement, and so to estimate the effect of the tuberculin *per se*.

This series were all patients at the State Tuberculosis Dispensary in Philadelphia (No. 21) and had been under observation continuously for a period of several years previously to being brought together in a special tuberculin class.[2] They had all had a prolonged cure at the State Sanatorium at Mont Alto (there were two cases previously treated at White Haven), and had returned to their homes on an average of from four to six months before the injections were started, during which time they had all been members of a tuberculosis class meeting every two weeks, in which their progress was carefully followed. They were selected from a group of several hundred patients on account of their physical condition and the nature of their lesions and because the entire course of their disease was known and they were all of sufficient intelligence and persistency of purpose to have been carrying out the most systematic treatment in their homes

[1] Read December 4, 1912.

[2] The practical work on these cases, namely, the giving of the tuberculin injections and the keeping of most complete and laborious records, was carried out by Dr. Hartz.

(which offered suitable facilities for this), and had proved themselves capable by their home-record charts of giving an intelligent account of their symptoms and condition under tuberculin. The number of cases, eleven, was purposely limited in order that they could be studied in greater detail and with the greatest care.

But the cardinal reason for their selection was that the cases all belonged to the chronic fibroid type, either moderately or far advanced, with little or no fever, and had already developed a fair resistance to the disease, yet in them there was observable a very gradual deterioration in weight and strength in spite of everything which could be done for them. This was observable by comparison of their condition at considerable intervals of time and was further indicated by the fact that they had all returned to work and been compelled by gradually failing health to give it up.

They had had the advantage of home and sanatorium treatment and a liberal diet, had lived under favorable hygienic conditions consistent with their station in life, and had been under constant supervision of physicians and nurses trained in tuberculosis work. The women were able to attend to their household duties, and the men had resumed their occupation on returning from the sanatorium, but had been compelled to abandon their work on account of physical incapacity which manifested itself sooner or later after resumption of their former occupations. They represented a class of fairly well-nourished individuals, most of them having measurably regained their average weight, and had felt themselves largely restored in health on leaving the sanatorium.

In other words, these patients under a most systematic and adequate regimen (which was in no way altered during the course of tuberculin) had remained either stationary in condition and weight for a long time, but considerably below par in general health, or in some instances they were slowly losing, each month or so showing a reduction in general efficiency and a gradual loss of weight.

It was therefore with a view of estimating the effect of the use

of tuberculin on the general health or functional betterment that the injections were undertaken: (1) As evidenced by increase in strength as measured by their ability to do more, that is, to return and remain at work; and (2) as evidenced by increased appetite and assimilation as indicated by increase in weight and the retention of this increase.

The patients kept a daily record of their temperature, which was recorded on a home chart provided for the purpose. Any untoward symptom, as increase in cough, expectoration, insomnia, nervousness, nausea, chills, fever, headache, or malaise, was also noted by the patient. Their observations were made twice daily.

The injections were given as a rule once a week. The weight of the patient, the dose of the injection, the pulse, temperature, and respiration were recorded immediately before the injection. Analysis of the patient's home chart as to any untoward symptoms resulting from the previous injection was during a succeeding period of five days carefully considered and then recorded on a special chart which was the permanent record of the progress of each case. These charts were kept at the dispensary. Each month the patient was reëxamined, recharted, and closely questioned as to his physical well-being, and the results were recorded on this permanent record in the patient's own statements. These observations were elicited without any undue suggestion of the examiner. The sputum of each patient when obtainable was examined every month.

Technique. The tuberculin used was the tubercle bacilli extract prepared after the method of S. G. Dixon.[1]

[1] Preparation of Tuberculin (Pennsylvania Health Bulletin, October, 1911). In the Medical News, October 19, 1889, attention was called to a morphologic condition of the tubercle bacillus which had never previously been observed. This change consisted in modifications of the organisms which suggested possible involution forms. These forms represented a difference in the degree of virulence which led to the subcutaneous injection of these somewhat devitalized organisms into tuberculous animals with a result of producing different degrees of immunity. Tubercle bacilli were cultivated in a number of tubes to a stage at which few or no ordinary rod-form bacilli could be microscopically found in the cultures, and inoculation into guinea-pigs and rabbits of these atypical and avirulent forms resulted in a short febrile stage with an enlargement of the lymphatic nodes located in the neighborhood of the point of injection. This condition, however, soon passed away and the animal recovered, and afterward exhibited a high degree of resistance against the effects of virulent tubercle bacilli. On further experimentation it was shown that by treating a mass of tubercle bacilli with ether and abstracting with a salt solution, a product was obtained that,

Each injection was in a separate aseptic syringe ready for use and put up not unlike diphtheria antitoxin. Ten different dilutions,

when injected into the animal economy, changed the condition of the tissue cells and thereby prevented the growth of the tubercle bacilli. The result of the work was first published in the Medical News of January 17, 1891, p. 87. This work was continued by Dr. Samuel G. Dixon until two methods of preparation had been published from time to time, as improved technique had been adopted resulting in the method hereinafter outlined.

Tubercle Bacilli Extract. This product is prepared from living organisms. The tubercle bacilli are grown on 4 per cent. glycerin veal broth for a period of from six to eight weeks at a temperature of 37.5° C. They are removed from the incubator and collected on hard filter paper. The filtrate of glycerin broth on which they are grown is discarded. An equal quantity, by weight, of tubercle bacilli of the bovine and human type is used. The mass of organisms is partially dried of the excess of moisture by placing between two sterile filter papers and afterward placed in a dish in the incubator for from twenty-four to forty-eight hours. The dried organisms are then washed in an excess of ether, which is allowed to act until it has removed all of the water and glycerin. The organisms are then subjected to an excess of fresh ether and washed in this for six hours to soften the wax of the bacilli. This fat separates so that it collects at the bottom of the vessel and is removed by a Pasteur pipette. After removal of the second addition of ether, the mass is allowed to dry until no ether odor is perceptible. After the mass of tubercle bacilli has been thoroughly dried, it is ground in a mortar and suspended in physiologic salt solution in the proportion of one part of the ground mass to five parts of an 8 per cent. salt solution. This suspension is carried in a shaking machine for from eight to ten hours and then allowed to stand for several days at room temperature. It is finally passed through impervious bacteria filters several times, and the filtrate is examined microscopically, bacteriologically and physiologically. Culture tests are made to determine its freedom from contaminating organisms, and guinea-pigs are inoculated to ascertain that it contains no living tubercle bacilli. One c.c. of this extract represents 0.5 gm. of the organisms, and is known as the stock solution from which serial dilutions are made. This solution is sterile, though a preservative of 0.5 per cent. of phenol (carbolic acid) is added to prevent any subsequent contamination.

The tubercle bacilli extract is supplied in syringes in the series of dilutions given in the following table so that the doses may be increased as deemed advisable.

Dilution No.	Amount of extract of tubercle bacilli contained (gram.)
1	0.001
2	0.01
3	0.02
4	0.03
5	0.04
6	0.05
7	0.06
8	0.07
9	0.08
10	0.09
11	0.10
12	0.11
13	0.12
14	0.13
15	0.14
16	0.15
17	0.16
18	0.17
19	0.18
20	0.19

It is suggested that in ordinary cases, the injection of each dilution be repeated at least five times before changing to the next number, or next stronger dilution. The physician

representing different strengths of tuberculin, were used. As many injections of one number (strength) as were consistent with the progress of the patient were given, usually three, sometimes as high as six, depending on the reaction induced. Every care was taken to avoid even slight reactions, and in this event the usual course was followed of omitting the next injection and repeating the same dose.

The weakest injection represented 0.001 mg. of tuberculin, the strongest 0.09 mg. The injections were given in the interscapular tissues of the back. The part was washed with ether, and as the syringe was in an aseptic state no other preparation was necessary. At no time was there any severe local reaction or any evidence of infection noted at the site of injection.

The dose should be small at the beginning, particularly so with the first six or eight injections, until tolerance is established, for if the increase be too rapid, reactions become more frequent and treatment must be discontinued until susceptibility wanes.

The time element is of great importance in the full course of treatment and should never be less than four months, and may with benefit be extended to a year or even longer. Reactions and the progress of the case under treatment must be considered in regard to length of treatment.

When the extract is given in suitable cases at regular intervals varying from periods of five to seven days and in appropriate doses, no harmful effects were noted.

Should a moderate or a severe reaction occur, instead of decreasing the dosage it is better to omit the following one or two injections until the patient's tolerance is raised, and then repeat the same dose.

should bear in mind the general condition of the patient during the administration of this treatment, and the same suggestions as are later outlined for the administration of the suspension of dead tubercle bacilli are applicable to this product. It is to be remembered, however, that the tubercle bacilli extract is toxic and must be used with caution. Small repeated doses injected into normal guinea-pigs cause no effect on their temperature, weight, or condition.

Case I.—W. O., carpenter, aged forty-six years, married; American. Diagnosis on admission, May 9, 1908, pulmonary tuberculosis, far advanced; prognosis unfavorable. Consolidation upper and middle lobes right side, with large cavity and heart displaced to right; consolidation left upper lobe. Active symptoms began three years previously with cough, loss of weight and strength. Average weight in health, 150 pounds. Height, 5 feet, 8 inches. Weight on admission to dispensary, 112 pounds. Weight on return from Mont Alto, December 15, 1909, after three months sojourn, 158 pounds. Patient on return began to cough and spit more and slowly lost weight and strength. He also began to have a little increase of temperature and, though up and about, was unable to work. Took the cure systematically, but did not improve. Weight dropped to 138 pounds.

Treatment and Results. Patient was under treatment seven months, following return from Mont Alto, before tuberculin injections were begun, July 12, 1910 (weight 138 pounds), and continued for eight months, during which time he received twenty-seven injections. At the end of three months he had gained 10 pounds, and his condition had improved so much that he was permitted to return to work on half time, and two months later to his regular work, eight hours a day. At the conclusion of his course of tuberculin he showed evidence locally of good arrest. He was practically without cough or other symptoms and working steadily.

November 27, 1912. Local condition very fibroid and inactive; few metallic, sticky rāles; general condition splendid; patient working at his regular employment right along (hours 7 A. M. to 5.30 P. M.) and during past June, July, and August from 7 A. M. to 8 P. M.). Some cough still and little expectoration on rising. Strength good. Weight 146½ pounds, 8½ pounds gain over weight at commencement of tuberculin, twenty-eight months ago.

Case II.—G. W., pearl-worker, aged forty-eight years, married; American. Diagnosis on admission, September 18, 1909, pulmonary tuberculosis, far advanced; prognosis unfavorable. Consolidation both upper lobes; cavity on left side. Active symptoms began nine years previously with hemoptysis and loss of weight and strength. Average weight in health, 130 pounds, Height, 5 feet, 4¾ inches. Weight on admission to dispensary, 116 pounds. Weight on leaving sanatorium (White Haven), where he had spent fourteen months in 1902, 135 pounds. Patient returned for a short while to work on leaving sanatorium, but has practically done nothing for ten years. He was coughing and spitting a great deal (2 ounces) on admission, was dyspneic, weak, and very thin, and was afebrile. Tubercle bacilli present. Weight, 116 pounds.

Treatment and Results. Tuberculin was begun October 2, 1909, and patient received in all thirty-five injections during a period of twelve months. Two months after first injection he had gained 6 pounds, felt much better, cough and expectoration were greatly decreased in frequency and severity, there was little sputum, and appetite and digestion were much improved. At the end of six months patient weighed 127 pounds, a gain of 11 pounds under tuberculin, and felt so much better that he decided to go to work and spent two months out of the city, during which time he worked regularly and supported himself. He returned, September 15, having gone down to 122 pounds but without the return of any serious symptoms; decided to continue his tuberculin treatment, about which he was enthusiastic. By February, 1911, at the close of tuberculin treatment, he had gained his former weight, 127½ pounds, and one year later, during which time he had been steadily at work, weighed 130 pounds.

November 15, 1912. Patient has been working right alone and feeling fine. For past fifteen weeks he has been working at Atlantic City as machine demonstrator (orange juice on Boardwalk) from 9 A.M. to 11.30 P.M. He did not miss a day. Weight is 123½ pounds, 7½ pounds more than at beginning of tuberculin treatment, thirty-seven months ago.

CASE III.—G. M., foreman, aged fifty years, married; American. Diagnosis on admission, October 27, 1909, pulmonary tuberculosis, moderately advanced; prognosis doubtful. Infiltration both upper lobes. Active symptoms began three years previously with pneumonia. Average weight in health, 160 pounds. Height, 5 feet, 8¼ inches. Weight on admission to dispensary, 133 pounds. Weight on return from Mont Alto, April, 1909, where he spent six months, 155 pounds. Patient still showed active lesions on both sides on return from Mont Alto and began slowly to deteriorate; was afebrile, but unable to work.

Treatment and Results. Tuberculin begun July 5, 1910, over a year after return from Mont Alto; weight, 143 pounds. Patient received in all twenty-nine injections in a period of eight months. Three months after first injection patient stated that he felt much better; was stronger, had more endurance and ambition, and had taken a position of watchman which was the first work he had done for past two years. On completion of injections, patient had returned to his regular work (foreman) and was working steadily and said that he felt better than he had at any time since illness began. Tubercle bacilli were present before and after tuberculin treatment. Physical signs those of an arrested lesion with evidence of chronic plastic pleuritis.

December 5, 1912. Patient is fifty-three years old and has felt very well, but is out of work and unable to get anything to do. He says he

feels better when he is working. He has a very slight cough and expectoration just after getting out of bed in the morning. There are a few small low-pitched crackling rāles on left (pleuritic); both upper lobes are fibroid. Weight is 140½ pounds, a loss of 2½ pounds over beginning of tuberculin, thirty months ago.

Case IV.—A. W. (sister of S. W.), bookbinder, aged twenty-one years; single; American. Diagnosis on admission, February 1, 1910, pulmonary tuberculosis, moderately advanced. Prognosis doubtful. Infiltration right upper lobe and left apex. Active symptoms began two years previously with hemoptysis. Patient was under treatment at dispensary two months before being sent to Mont Alto, slowly losing ground. Average weight in health, 90 pounds. Height, 5 feet. Weight on admission to dispensary, 86½ pounds, in February, 1910. Weight on return from Mont Alto in July, 1910, where she spent three months, 91¼ pounds. Patient was observed for one month after leaving Mont Alto, during which time she felt weak, tired easily, and was unable to work; was afebrile.

Treatment and Results. Tuberculin was begun July 2, 1910, and patient received a total of thirty-six injections during a period of nine months. After having been on tuberculin for two months, patient felt so much better and stronger that she was permitted to return to work on half time. Her cough had entirely left her. Before the completion of her tuberculin course, she had returned to her regular work and could be seen by the nurse only on Sundays as she was out at her place of employment during the whole of each day. One year after last injection patient states that she feels as well now as at any time previous to her illness, and can do as much work. She is regularly employed daily and has been so right along.

November 27, 1912. Patient feeling fine; no cough or other symptoms; working regularly (hours 8.30 A.M. to 5 P.M.); has not missed a day. Signs of arrested lesion. Weight 89¾ pounds, a loss of 1½ pounds over weight at commencement of tuberculin, twenty-nine months ago.

Case V.—S. W. (sister of A. W.), school girl, aged twelve years; American. Diagnosis on admission, February 17, 1910, marked tuberculous adenitis, cervical and axillary glands; probable bronchial glands. No pulmonary localization. Active symptoms began with enlargement of cervical and axillary glands and cough one year previously. Average weight in health unknown. Height, 4 feet, 9¼ inches. Weight on admission to dispensary, 75 pounds. This was distinctly a glandular case and the glands on both sides of neck were becoming rapidly larger (as big as hen's eggs), tenser and most tender. There were large glands in both axillæ.

Treatment and Results. Tuberculin was begun March 15, 1910, and patient received thirty injections during a period of eight months. Following the first few injections of tuberculin the glands became perceptibly less tender and after several months were greatly reduced in size and entirely painless; at the conclusion of the treatment the glands were barely palpable. Patient gained steadily in weight from 75½ pounds on first injection to 85 pounds on date of last tuberculin injection; had returned to school and was apparently in normal physical health.

November 27, 1912. Patient is feeling well and has attended school regularly since. Examination of chest is negative. Glands in neck are palpable (about the size of a pea), but give her no trouble. Weight is 91¼ pounds, 15¾ pounds gain over weight at commencement of tuberculin, thirty-two months ago.

Case VI.—Samuel W., tailor, aged thirty-five years, married; Russian. Diagnosis on admission, February 1, 1908, pulmonary tuberculosis, moderately advanced; prognosis doubtful. Consolidation right upper lobe. Active symptoms began five years previously with influenza and hemoptysis. Average weight in health, 145 pounds. Height, 5 feet, 6 inches. Weight on admission to dispensary, 129 pounds. Weight on admission to Mont Alto, 117 pounds. Under dispensary treatment for six months, but failing steadily. Weight on return from Mont Alto, December, 1909, where he spent six months, 127 pounds. (Patient suffered while at Mont Alto with chronic gastritis and vomiting after meals.) Under treatment three months following return, but was slowly deteriorating; afebrile but unable to work.

Treatment and Results. Tuberculin begun March 1, 1910 (weight 117¼ pounds), and patient received in all thirty-eight injections during a period of eleven months. The sputum for one year preceding injections showed the presence of tubercle bacilli. Since tuberculin injections were begun, no bacilli could be demonstrated in the sputum; three months of tuberculin greatly improved patient's condition and he was able to resume his work for the first time in three years. After the last injection the following information was elicited in the patient's own statements: "For past three years I was not able to attend to my work at tailoring and carrying bundles of clothes. Three months after first injection, I commenced to work at my trade and have been doing so for past seven months. I work ten hours daily and feel better in every respect. The cough, expectoration, 'wind,' appetite, digestion, and sleep are very much improved, and I become less exhausted at work." Ten months after last injection patient is in good condition; works daily; lesion in the lung shows no activity.

November 27, 1912. Patient working on full time (hours 7.30 A.M. to 5.30 P.M.); supporting family of eight; feeling well and strong. Both upper lobes fibroid; weight 121 pounds, a gain of $3\frac{3}{4}$ pounds over weight at commencement of tuberculin, thirty-three months ago.

CASE VII.—M. D., aged fifteen years, single; American; occupation housework. Diagnosis on admission, July 6, 1908, pulmonary tuberculosis, moderately advanced; prognosis doubtful. Infiltration right upper lobe and left apex. Active symptoms began two years previously with cough, and loss of weight and strength. Average weight in health, 108 pounds. Height, 5 feet, 3 inches. Weight on admission to dispensary, 102 pounds. Weight on leaving sanatorium (White Haven), one month previously, where she had spent five months, $107\frac{3}{4}$ pounds. Patient was under systematic treatment for seven months, slowly deteriorating, but afebrile; tires easily and unable to work. Weight slowly went down to 99 pounds.

Treatment and Results. Tuberculin was begun February 1, 1910, and patient received a total of fifteen injections in a period of six months. As she developed a hypersensibility, the injections were given at longer intervals. Her weight two months after beginning tuberculin treatment reached 109 pounds, which was approximately retained until the very hot weather of July and August. Weight at last injection in August was $105\frac{1}{2}$ pounds. Patient states that she suffers from the hot weather, but cough and expectoration are much improved and she feels much stronger. She helps her mother about the house. Tubercle bacilli were found previous to the tuberculin, but not during or after the treatment.

December 3, 1912. Patient in fine condition, working regularly in a department store. No cough or other symptoms. Weight, 130 pounds, 31 pounds more than weight at beginning of tuberculin treatment, thirty-four months ago.

CASE VIII.—W. B., weaver, aged forty years, single; English. Diagnosis on admission, December 16, 1908, pulmonary tuberculosis, far advanced; prognosis doubtful. Infiltration upper and middle lobes right side; consolidation with cavitation, left upper lobe. Active symptoms began two years previously with severe hemorrhage. Previous to admission patient had spent three months in tuberculosis wards, Philadelphia General Hospital. He was six months under treatment at dispensary before he was sent to Mont Alto, losing ground steadily. Average weight in health, 130 pounds. Height, 5 feet, $9\frac{1}{4}$ inches. Weight on admission to dispensary, $110\frac{1}{4}$ pounds in December, 1908. For one year after his return from Mont Alto he was under most systematic treatment. He attempted to return to work but could not stick at it.

He was slowly failing; had no appetite, poor digestion, and slight fever; cough and expectoration troublesome.

Treatment and Results. Tuberculin was begun February 15, 1910; patient received thirty-nine injections during a period of twelve months. Four months of tuberculin treatment found the patient with a net gain of $10\frac{1}{2}$ pounds from the time of first injection; cough and expectoration much less and great gain in general health; entirely afebrile. Patient went to work four months after beginning tuberculin, and continued at his work as night watchman throughout entire tuberculin treatment, reporting regularly once a week, except toward the end when his earning capacity having increased and his general health being so much better, he began to consume alcohol in varying amounts, became irregular in his habits, and finally was lost sight of. He died April, 1912.

Case IX.—E. S., tailoress, married; Russian. Diagnosis on admission, January 27, 1909, pulmonary tuberculosis, moderately advanced; prognosis fair. Consolidation right upper lobe. Active symptoms began two years previously with attack of influenza and hemorrhage. Average weight in health, 135 pounds. Height, 5 feet, $3\frac{1}{2}$ inches. Weight on admission to dispensary, January, 1909, 114 pounds. Weight after return from Mont Alto, August 25, 1910, where she staid eight months, $141\frac{3}{4}$ pounds. She returned to work, took cold, and began to lose weight; had some cough and expectoration, slight fever; was unable to continue at work.

Treatment and Results. Tuberculin begun October 4, 1910; weight, 138 pounds; patient received a total of forty-two injections in a period of twelve months, the treatment having been started at Mont Alto. Patient returned to work (three days a week), November 22, 1910, and full time in December, 1910. Weight on conclusion of tuberculin treatment, five months after return from Mont Alto and on full-time work, $140\frac{1}{4}$ pounds. Fourteen months after last injection, patient is working regularly daily. Cough and expectoration are much less than formerly; patient is stronger and feels better in every way. Lesion involving right upper lobe is in an arrested stage. Tubercle bacilli were present before and after tuberculin treatment.

November 27, 1912. Patient has been working steadily ever since in tailor-shop, supporting self and two children. She still has some cough and expectoration and rāles on right side. Weight is 130 pounds, a loss of 8 pounds over weight at commencement of tuberculin twenty-five months ago.

Case X.—C. McG., housewife, aged forty-one years, married; Irish. Diagnosis on admission, Macrh 16, 1909, pulmonary tuberculosis, far advanced; prognosis unfavorable. Consolidation upper and middle

lobes, right side and left apex. Active symptoms began four years previously with pleurisy. Patient was at White Haven five months previous to admission; returned April, 1908. Average weight in health, 155 pounds. Height, 5 feet, 7 inches. Weight on admission to dispensary, 142½ pounds. Weight on return from Mont Alto in July, 1910, where she spent four months, 145 pounds. Patient was treated one year at dispensary before being sent to Mont Alto, during which time her weight remained stationary and she was afebrile, but suffered with several attacks of blood-spitting. No tuberculin was given during this time.

Treatment and Results. At Mont Alto patient received a course of eleven injections of tuberculin, was free from blood-spitting and said she felt much improved. She asked to have her tuberculin continued and injections were resumed at dispensary. She received eleven more, or a total of twenty-two injections in eight months. At the end of this course patient weighed 142½ pounds, felt much better and stronger, cough and expectoration were less, and she resumed her work. Six months later patient was in good condition and doing her own housework. Patient died August, 1911.

Case XI.—G. S., ironworker, aged forty-two years, married; American. Diagnosis on admission, February 11, 1909, pulmonary tuberculosis, moderately advanced; prognosis doubtful. Consolidation both upper lobes. Active symptoms began five months previously with cough, hemoptysis and loss of weight. Average weight in health, 160 pounds. Height, 5 feet, 7 inches. Weight on admission to dispensary, 150 pounds. Weight on return from Mont Alto, August 11, 1909, where he spent five months, 163¾ pounds. This case was complicated by chronic tuberculous laryngitis, and the patient did not improve much at the sanatorium. He had much cough and expectoration on return (filled two sputum boxes at night) and was unable to work. Tubercle bacilli were constantly present; no fever.

Treatment and Results. Tuberculin treatment was begun February 15, 1910, six months after return from Mont Alto. Patient's weight was 157 pounds; he received twelve injections during a period of four months. Patient was much improved in general health, cough and expectoration noticeably decreased, and he left so well that he insisted on returning to work, securing a position as watchman. One year after last injection patient was working steadily and feeling fine.

November 27, 1912. Patient has been working for some months for Adams Express Company, doing trucking of heavy freight, much heavy lifting (hours from 6.30 p.m. to 7 a.m.), twelve hours of heavy night work. Three weeks ago packing-case slipped and fell on shoulder,

breaking the left arm. Patient has been worse since his injury, coughing and spitting more and suffered much pain in hospital. He lost 10 pounds in three weeks; weight previous to accident 150 pounds, a loss of 7 pounds over weight at beginning of tuberculin treatment thirty-nine months ago.

Conclusions. The conclusions which we feel justified in drawing from a thorough familiarity with the course of health in the cases above described for a long period of time is as follows:

1. These cases were all (with one exception, the glandular case) of an advanced chronic fibroid type, which had under treatment shown a fairly good resistance, but which in spite of everything which could be done for them, were unable to work and slowly deteriorating in health, weight, and strength; this deterioration was at least checked in every case while tuberculin was being used.

2. Further, every patient was distinctly improved in health and was able to resume and remain at regular work during or following the course of tuberculin.

3. We recognize the fallacy of attributing too much to any one factor or therapeutic measure in chronic diseases generally, and in tuberculosis particularly. In emphasizing the fact that these patients were so improved and were able to return and continue at work, we do not mean to infer that such results are not seen in chronic advanced tuberculosis untreated by tuberculin; but we do argue that from the previous course of health in these particular cases under most systematic treatment, the tuberculin at least seemed to be the sole added factor responsible for the improvement.

4. The tuberculin apparently cured the patient with glandular tuberculosis.

5. Tubercle bacilli disappeared from the sputum in several of the cases during the course of treatment, but the majority of the patients still showed the presence of tubercle bacilli on the completion of the full course of injections.

6. The local condition in the lungs showed a distinct improvement as evidenced by the disappearance of rales.

7. In advanced chronic tuberculosis, when there is fair resistance and little or no fever, tuberculin is unqualifiedly a valuable therapeutic measure, or at least proved so in the above series.

8. The reactions with Dixon's tuberculin (tubercle bacilli extract) were uniformly mild and without unfavorable influence on the course of treatment.

9. All the different preparations of tuberculin represent an effort to retain the favorable toxins of the bacillus and to eliminate those which are deleterious to the human organism; and we believe that for general use at least, particularly in ambulatory cases, it is safer and better to select a preparation of mild potency and toxicity, and that in the so-called Dixon's fluid we have a safe and effective tuberculin of this type.

DISCUSSION

Dr. H. J. Hartz: I should like to emphasize some of the points that were brought out in the paper. The class of cases selected were admirably suited for the study of the effect of tuberculin therapy. All were patients that were under observation for long periods of time, have had the advantages of sanatoria, dispensary, and home treatment, had been members of tuberculosis classes, yet withal, had failed to improve and were slowly retrogressing. It is only fair to assume that the marked improvement noticed in these cases can only be attributed to the tuberculin, since the routine treatment of the patient was in no way altered during the course of the tuberculin injections. The enthusiasm displayed by the patients during the course of treatment was noteworthy, often discussing the effects as they were sitting in turn for the injections, and were elated over the results. The functional ability of the patients was increased, enabling them to return and to stay at work, thus becoming less dependent upon other members of the family. Aside from a few reactions and untoward symptoms that developed if the dose of the injection was increased too rapidly, the general course of the treatment was favorable in every instance.

Dr. James Tyson: I should like to say a word in commendation of the method in which this paper is prepared. The evidence as to the actual effect of the tuberculin treatment is the clearest I have yet met.

The cases are limited in number, the duration and method of treatment are well stated, and the results are definitely indicated. The paper is, to my mind, a very satisfactory and encouraging one so far as this line of treatment is concerned. I assume that in the cases in which the glandular condition is spoken of, the condition is associated with pulmonary tuberculosis.

Dr. John B. Deaver: It may seem out of place for a surgeon to rise to discuss the treatment of tuberculosis by tuberculin. I am, however, so much pleased with this paper, and have for so long a time advocated the treatment of many surgical lesions with the tuberculin, that I am heartily in accord with what Dr. Francine and his associate, Dr. Hartz, have said. I am glad to see that the doctors are waking up, and I feel that tuberculosis surely will be cured, to my mind, along purely scientific lines.

Dr. S. Solis Cohen: I did not catch from the paper, and this may have been due to my coming in late, whether or not tuberculin injections were used in febrile cases or whether the cases were all afebrile; also whether during febrile reaction the tuberculin injections were intermitted.

I should also like to ask Dr. Francine upon what signs or symptoms or principles the injections were discontinued; in other words, whether it was determined arbitrarily, that fifteen or some other number should be the number of injections, or whether certain changes in the patient's condition indicated the time to cease treatment.

I have had some experience, extending now over a large number of years, in the treatment of cases of pulmonary tuberculosis of different types with tuberculin in various forms; and while it has a certain degree of usefulness at times, I cannot say that I am very enthusiastic about it. I do see in certain selected cases, as the reader of the paper has well pointed out, an additional impetus to recovery. When a patient has been improving but, reversing the prophet's injunction, ceases to do well, and begins to do ill, then, perhaps, by timely injection of tuberculin for a limited period we can arrest the retrograde movement or even convert it into progress. Apart from this, I have never found any remarkable results. I have not found continued improvement from tuberculin, except in a number of cases so few in proportion to the whole number in which I have attempted the treatment, that I have been in doubt whether it were really due to the tuberculin or not. Still, in some of these few, the result was worth while.

I should like to call the attention of the Fellows of the College to two forms of tuberculin which may be considered along with the others—Dixon's I have no personal experience with, but this paper illustrates its value very well. The others I have used. One that is not generally

known is "tuberculinum purum" advocated by Gabrilowitch and Stenius. From this are removed by extraction the harmful albumoses to which the untoward symptoms of Koch's early preparation were attributed. The reactions are not febrile, at least, such is the general reports from those who have used it, and such is my own observation. Then, secondly, there is Latham's method of administration by the mouth, instead of hypodermically. Latham used "tuberculin residue," and to follow up his observations in his own way I used the same thing. It is commonly termed "T. R."—but without reference to any notable individual. The tuberculin residue may be administered by the mouth in an initial dose of 0.00001 mg., increased if necessary to as much as 0.01 mg. Some have used 1 mg. I have never gone beyond 0.01 mg., and rarely beyond 0.001 mg. I have seen distinct reactions from 0.00005 mg. The general rules of cautious increase apply here as everywhere.

There are various methods of giving tuberculin by the mouth. Latham prefers to give it with horse serum, as he thinks that thus the absorption is better. I have not been able to obtain horse serum readily in sufficient quantities, and find that beef juice or skimmed and diluted milk or whey answers just as well. Certain manufacturers have now, at my request, undertaken to put it up in tablet form in various strengths, so that the administration may be conveniently gauged and regulated, small doses gradually increased being used just as in the ordinary dilutions for hypodermic injection. I have seen just as good results from the use of tuberculin in this way as from its hypodermic use, and among the few instances of remarkable improvement I have alluded to are several in which patients have thus taken tuberculin residue by the mouth over a period of many months—in one case for nearly two years. I find it the most convenient method with ambulant patients, and the most readily controlled when prolonged administration seems to be indicated; or when the administration is intermitted and renewed from time to time.

Dr. George E. de Schweinitz: While not entirely germane to the subject of pulmonary tuberculosis, the Chair desires to confirm the high value which Dr. Francine has placed upon the proper use of tuberculin as a therapeutic agent in so far as various tuberculous lesions of the eye are concerned, particularly those which affect the uveal tract, the sclera and the cornea, and since this remedy has come into vogue, these ocular lesions have yielded much more readily than they did under other and older therapeutic measures.

Dr. Francine closes: The glandular involvement was a tuberculosis of the glands uncomplicated by any pulmonary localization, and these glands were on the point of breaking down. They were red, inflamed,

very much enlarged, and very painful. This case and her sister represented a very early infection, probably in infancy, as their parents were both tuberculous. As one so often sees in this type of infection, the active symptoms do not develop until puberty or later. There is a long period of apparent latency. Further, where the glands are particularly attacked, or where the localization of the active process is in the glands, the lungs frequently escape (at least for a long time), and when involved show a very sluggish, low-grade process. This is not without exceptions, of course, but conforms at least to my experience. The same thing is seen where the active localization is in the bones, as in Pott's or spinal disease in children. These are particularly favorable cases for tuberculin.

This view, too, conforms with the very interesting statement of Dr. de Schweinitz, as to the effect of tuberculin where there is localization in the eye.

In regard to the length of treatment, no definite rule can be laid down, any more than a hard and fast system for the increase in dose or frequency of injections. Every case must, of course, be treated individually, and the injections must be carefully adapted to each patient's resistance, general health, improvement, etc. So long as there is improvement and so long as the patient feels better after the injections, one would be naturally disposed to continue the course, though personally, I think a period of a year should be the outside limit. Then wait to see how permanent and what degree of resistance the patient has developed, and should this after a reasonable period begin to wane, there is no objection to repeating the course later on.

I have never used tuberculin by the stomach or the tuberculin purum to which Dr. Cohen refers. Personally, I am inclined to believe that all tuberculins have very much the same effect, and presumably one may use with benefit any of the standard preparations, provided you use the stronger and more toxic preparations in small doses and over suitable periods of time, and always with regard to the features of the individual cases. Of course, I prefer a tuberculin of mild toxicity as mentioned above, which I think our experience has proved Dixon's fluid to be. Because a tuberculin is of mild toxicity does not imply that it is less effective therapeutically. On the contrary, we have all learned the greater wisdom and efficiency of small doses. In this series we used Dixon's tuberculin which, of course, acted well in these cases. The cases were all practically afebrile, and had developed a pretty fair resistance (through prolonged cure), and were in pretty fair general health.

Dixon's tuberculin seemed to be just what was needed in these cases to give them the added resistance to tide them over their period of waning resistance, and enable nature to reassert itself. For, of course,

in the last analysis tuberculin is only a life belt enabling the patient to get to shore only if he has resistance and recuperative powers of his own sufficient to do this. For this reason, I feel that the subsequent death of two of our cases does not in any way militate against the position we take, because these two cases improved equally with the rest, but their increased resistance was not sufficient to overcome the progress of the disease and associated extraneous circumstances (see case histories).

Of course, it is well known that the more severe reactions of fever are not suitable for tuberculin. Upon the advent of fever in the course of tuberculin treatment, the injections must, of course, be modified or discontinued, as outlined in the text of our paper.

ACUTE UNILATERAL NEPHRITIS, WITH REPORT OF A CASE[1]

By DAVID RIESMAN, M.D., and GEORGE P. MÜLLER, M.D.

Synopsis of authors' case: Woman, aged twenty-two years, seized with sudden pain in left loin, radiating forward into left hypochondrium; rigidity of lumbar muscles with tenderness in costovertebral angle; fever; rapid pulse; leukocytosis; in urine albumin, hyaline and granular casts, red blood cells, and pus cells. Patient moderately septic; nephrectomy; kidney the seat of disseminated, suppurative nephritis. Patient made a good recovery, although some weeks later it was necessary to remove submaxillary gland for calculous inflammation.

Pathological and clinical studies have caused the belief to prevail that acute hematogenous inflammation of the kidney is always bilateral. This is, however, not the case; but exceptions to the rule are so rare and so little understood that reports of single instances are not without value. Acute unilateral inflammations differ from the commoner types of acute nephritis in that they are of an interstitial character; more inflammatory than degenerative, and with a strong tendency to the formation of abscesses. Acute bilateral interstitial inflammation has long been known. It was found by Councilman[2] in scarlet fever, and has been seen in erysipelas, osteomyelitis, endocarditis, and pyemia. The infection in these cases is conveyed to the kidney by

[1] Read November 6, 1912.

[2] Transactions of Association of American Physicians, 1898, vol. xiii.

the blood stream. Suppurative inflammation, pyelitis and pyelonephritis, is of course common in association with stone and tuberculosis. It is usually considered to be due to ascending inflammation, although this is by no means clearly established. In addition there is a group of borderline cases, as for instance the pyelitis and pyelonephritis of pregnancy and the puerperium, in which it is difficult to say how the inflammation reaches the kidney. We are inclined to believe that even in these it is more likely hematogenous than an extension from below upward.

The particular type of unilateral nephritis to which we wish to refer in this communication is that which attacks a person apparently well, often without warning, and with an unusually acute onset. In some patients the progress of the disease may be so rapid that in a few hours extreme prostration is observed, while in others the course may be protracted so that several days elapse before marked septic symptoms are noted. The patient complains of tense, throbbing pain in the loin, often radiating into the anterior aspect of the abdomen. There is tenderness over the same area but most marked at the costovertebral angle, and this is accompanied by rigidity of all of the muscles of the loin and of the oblique muscles of the abdominal wall. There is fever of variable degree with a correspondingly rapid pulse and a moderate leukocytosis. The urine is rather scanty in amount, of high color and turbid in appearance. It contains a considerable amount of albumin, and the sediment consists largely of casts, red blood corpuscles, and pus cells. Certain general manifestations such as headache, nausea and vomiting, delirium, and even symptoms of uremia may appear, and the patient looks and is extremely toxic.

In 1895, Weir[1] and Woodward[2] each reported cases of nephrectomy for unilateral suppurative nephritis but the present surgical treatment of this affection is mostly due to the able teachings of Brewer,[3] who has persistently called attention to

[1] Medical Record, 1894, vol. xlvi, p. 325.

[2] Annals of Surgery, 1895, vol. xxi, p. 568.

[3] Surg. Gynec. and Obst., 1906, vol. ii, p. 485; Ibid., 1908, vol. vii, p. 699; Jour. Med. Soc., New Jersey, 1909, vol. vi, p. 61; Yale Med. Jour., 1911, vol. xvii, p. 237.

the subject and in his latest contribution reports an experience of 14 cases of the acute type and about a dozen of the mild type. Cobb[1] collected 8 cases from the surgical wards of the Massachusetts General Hospital, all of them operated upon since 1902. There are a few other instances recorded in the literature, and the number is increasing sufficiently so that we shall soon become familiar with the affection and approach its diagnosis and treatment with more certainty and confidence.

The case that we have observed and to which we have made brief reference is as follows:

Mrs. R. A. K., aged twenty-two years, had measles, mumps, and scarlet fever when a child; one year ago she had a severe attack of typhoid fever complicated with hemorrhages but convalescence was not otherwise interrupted and she was quite well during the summer of 1910. Three months ago she was married. During the early part of December, 1910, she suffered for two weeks from general aches and pains supposedly influenzal in origin, after which a vague pain persisted in the left loin, and she herself believed that some swelling was present, but her physician did not notice anything. On December 23, she rode 14 miles into the country in an automobile, the day being exceedingly cold. At 3 A. M. on December 24, she awakened nauseated and had an attack of vomiting; some hours later moderate pain in the left loin was complained of, and there was tenderness and rigidity; the temperature rose to 102° F. and the pulse to 120; the leukocytes on this day were counted and found to be 7000, but two days later they had risen to 13,000. Daily examinations of the urine were made and showed it to be highly colored and containing albumin and casts but no pus cells. The attack gradually subsided, pain and tenderness disappeared, but some rigidity persisted. On December 30, she insisted on getting out of bed, following which act the symptoms recurred, and about this time it was noticed that pus was present in the urine. We saw her first on December 31, 1910, at her home in the country.

We found a well nourished, rather pale young woman, complaining of pain in the left loin and left hypochondrium, radiating at times downward to the groin and sometimes to the back. Tenderness to pressure existed at the costovertebral angle and extended forward to the crest of the ilium. There was distinct rigidity of the muscles in the loin and a sense of fulness to palpation, although no tumor could be detected. There was

[1] Annals of Surgery, 1908, vol. xlviii, p. 680.

fever and a rapid pulse and a moderate leukocytosis, and urine analysis showed some albumin with hyaline and granular casts, erythrocytes, and pus cells in the sediment. The patient was not particularly prostrated although she looked septic. We made a positive diagnosis of a unilateral infection of the kidney. As she was a woman with abundant vitality and energy, we believed it would be safe to temporize a few days and await developments. Accordingly, hot fomentation were ordered for the kidney region, and urotropin and an abundance of water, liquid diet, and purgation were prescribed. During the next ten days the patient varied in her condition, sometimes being on the verge of marked improvement, but again having a relapse to the condition just mentioned. Finally, as the temperature continued around 102°, and as her general appearance indicated an increasing septic state, and as some delirium was manifested, an operation was deemed advisable. The symptoms were always unilateral and always located in the region of the left kidney. She was admitted to St. Agnes' Hospital, January 11, 1911. During the few hours before operation, the temperature varied from 100° to 102°; the pulse from 84 to 100.

The operation was performed January 11, 1911, under ether anesthesia. The patient was placed in the usual lateral position over a kidney pillow and a curved incision made downward and inward from near the costovertebral angle. Upon opening the deep fascia, the perinephric fat was found infiltrated and inflamed, bleeding with great readiness. Upon further exploration of the fat in the region of the kidney, an abscess was encoutered containing 3 or 4 ounces of thick pus. The kidney was exposed and delivered with some difficulty and was found to be the seat of a diffuse, suppurative process. Accordingly, nephrectomy was performed and the wound packed with gauze and drained with a rubber tube. Partial closure of the wound was effected.

Examination of the specimen was made by Dr. Aller G. Ellis, Dr. John Speese, and ourselves, and the examination of the pus by Dr. Randle C. Rosenberger. The reports are as follows:

Macroscopic Description. The kidney was about one and one-half times the normal size, intensely red and congested, with purple blotches of hemorrhage beneath the capsule. Near the lower pole a ragged opening existed with evidence of necrosis at its edges. In three other places rents were found, evidently caused by trauma during operation. It was afterward discovered that at these places hemorrhagic infarction existed. Upon section of the kidney the entire organ was seen to be intensely congested, especially the cortical portion, and to be the seat of numerous miliary abscesses. In the lower half, a large wedge-shaped yellow area communicated with the opening before mentioned and which

had probably been made at operation. The apex of the wedge represented the apex of a pyramid. One other such area, but somewhat smaller in size, was present. At the upper pole a round, orange-colored mass about 4 mm. in diameter was discovered which was thought to be tuberculous, but wes discovered afterward upon microscopic examination to be a hypernephroma.

Microscopic Description. Sections show a hemorrhagic, fibrinocellular exudate on the surface of the renal capsule. At several points a large amount of hemorrhage is present and exists as an infarct. The capsular vessels are dilated, and a diffuse leukocytic infiltration begins beneath the capsule and extends downward through the cortex and medulla. Many of the leukocytes are of the polynuclear form. The renal epithelium shows degenerative changes and cloudy swelling with numerous casts. The stroma in some areas is decidedly fibrous.

There was no evidence of tuberculosis in any of the sections examined by Dr. Speese, Dr. Ellis, or ourselves. In the specimen submitted to Dr. Ellis a small hypernephroma was discovered with extensive connective tissue formation.

Post-operative History. The patient reacted from the shock in a few hours, after which time the temperature gradually rose until on the fourth day it was 103° F., after which time it slowly declined to the normal, which was reached on the sixth day after operation. The pulse remained high, rapid, and weak for several days after operation. The patient commenced to pass urine shortly after the operation, and during the first twenty-four hours 17 ounces were passed. During the second twenty-four hours 41 ounces, and during the third twenty-four hours 57 ounces were voided. The amount of urine during the first ten days after operation varied from 33 to 57 ounces, and averaged 44 ounces, and during the last ten days of her stay in the hospital it varied from 31 to 46 ounces, and averaged 39 ounces. At first there was abundant albumin with casts, pus cells, and erythrocytes, but the sediment gradually lessened and for the last week examination was negative for albumin, casts, and pus. The last urine analysis showed a specific gravity of 1010. The gauze packing was removed from the wound during the second week, but considerable wound troubles were experienced which protracted convalescence. A bacteriologic examination of the pus in the kidney removed at operation and later of the wound discharge revealed the Micrococcus pyogenes aureus, and from the culture a vaccine was made by Dr. Rosenberger and administerred seveal times to the patient. The examination of the blood showed hemoglobin 38 per cent. on the day after operation, and from this it hardly varied until February 14, when it began to rise steadily. The first count of red blood cells made

four days after operation revealed 3,200,000, and the count varied but little from this until February 14, when it showed 3,940,000, after which continued improvement took place. The leukocytes varied, depending on the condition of the wound, but averaged about 13,000 during her stay in the hospital.

The patient was discharged February 25, 1911, forty-four days after admission, with a small sinus rapidly closing. Her general health was excellent, although some weakness persisted. The sinus healed entirely in one week. About six weeks later, the patient complained of swelling, pain, and tenderness beneath the right lower jaw and noticed some salivation with a foul odor and salty taste in the mouth. An examination revealed a swelling of the right submaxillary salivary gland, densely hard in character, not acutely inflammatory but somewhat tender to pressure. Pressure caused pus to exude from the duct of Wharton into the mouth. An *x*-ray plate made by Dr. Pancoast showed the presence of a calculus.

Under ether anesthesia a two-inch incision was made beneath the jaw, and the submaxillary salivary gland removed. It contained an oval calculus about 12 mm. in length. Since this time the patient has been perfectly well, gaining in weight and strength, and frequent urine analyses have shown nothing abnormal. Six months ago she became pregnant; nothing unusual happened until October 27, when a miscarriage occurred. The patient suffered no ill effects from this, however. On November 1, she was passing 40 ounces of urine in twenty-four hours with a specific gravity of 1015, and showing nothing abnormal to chemical or microscopic examination.

Etiology. The kidney in unilateral nephritis may become infected in one of three ways:

1. *By an Ascending or Urogenous Infection.* This is much less common than was once held, and Sampson's[1] well-known paper published in 1903 states the case in the following words: "The reflux of urine from the bladder into the ureters may be considered an etiological factor in the causation and maintenance of renal infection only when the intravesical portion of the ureter is diseased, thus impairing its function, or when some ureteral abnormality exists." It will thus be seen that the kidney is protected from ascending invasions by the downward current of urine and the physical condition of the healthy

[1] Johns Hopkins Hospital Bulletin, 1903, vol. xiv. p. 334.

ureteral orifice. Of course, infection may be transmitted to the pelvis of the kidney by an unclean ureteral catheterization.

2. *Lymphatic Infection.* A local spread of infection from the intestinal tract or from the lower urinary organs by way of the lymphatics is not common but does sometimes occur. Some interesting experimental work has been published recently by Kunita,[1] showing the way in which the lymphatics of the ureter communicate with those of the perinephric tissue and kidney, and by Franke,[2] who found that the ascending colon and cecum were connected by a chain of lymphatics with the right kidney, but he was unable to find such connection in the case of the left kidney.

3. *Hematogenous Infection.* This is the common mode. It is well known that microörganisms frequently pass through the kidney without injuring it in so far as we know. The urine swarms with typhoid bacilli in many cases of typhoid fever, and one frequently observes a colon bacilluria; although in neither case are symptoms necessarily present indicative of kidney infection. There has been some experimental work published to the conclusion that the passage of microörganisms through the kidney is always attended by some damage histologically, although clinically no trouble may be appreciable. It has also long been known that by experimentally tying the ureter, bruising the kidney, and injecting cultures of staphylococci into the veins, the kidneys will become infected. These infections point the way to an understanding of the workings of well-known clinical conditions as factors in the etiology of pyelonephritis. Most cases of pyelonephritis are probably unilateral in the beginning, and, theoretically, a source of infection must exist somewhere in the body, although it is not always easy to ascertain the source. In view of the fact that the colon bacillus is the most frequent cause of urinary infections we may infer that intestinal stasis, intestinal ulceration, and severe and long-continued constipation act as predisposing factors. Previous

[1] Quoted by Stewart, Univ. of Penna. Med. Bulletin, 1910, vol. xxiii, p. 233.
[2] Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1911, vol. xxii, p. 623.

infections, such as furuncle, tonsillitis, tooth abscess, or any form of peripheral suppuration may predispose to pyelonephritis. The bladder, the prostate gland, and the uterus and its appendages may also act as portals of entry, and certain general infections, of which typhoid fever and influenza are the most prominent examples, may contribute to hematogenous infection of the kidney.

In addition to the entrance of infection, there must be some cause, traumatic or otherwise, operating to reduce the resistance of the renal tissue. This likewise is not always easily determined. Many of the cases reported as acute unilateral pyelonephritis with suppuration have occurred suddenly in the apparently normal kidney of persons previously well, and the infecting agent was usually the colon bacillus or the staphylococcus aureus. It may be that these organisms were highly virulent or in excessive number, or it may be that some unrecognized abnormality of the kidney was present. Several years ago, Kidd[1] suggested that as the majority of cases occur in women and involve the right kidney, undue mobility of the organ may be a predisposing factor, and Cotton[2] reports two cases in which there was a definite displacement of the infected (right) kidney. The microorganisms usually found in pyelonephritis are the Bacillus coli communis, Micrococcus aureus, streptococcus, and the typhoid bacillus, although a large number of different organisms have been isolated in different cases. The great majority of cases have occurred in women and the only explanation would seem to be more frequent displacement of the right kidney and the greater frequency of intestinal stasis in that sex.

Recently unilateral kidney hemorrhage of apparently unknown origin and sometimes called essential hematuria, has been placed in the group which we are discussing and considered to be of bacterial origin. Thus, Gaudiani[3] concludes that with the exception of early tuberculosis, lithiasis, neoplasms, torsion of the pedicle or Bright's disease, all cases described as hemorrhagic nephralgia are cases of unilateral nephritis and are the result of

[1] Urinary Surgery, 1910.
[2] Annals of Surgery, 1911, vol. liv. p. 577.
[3] Folia Urologica, 1908–09, vol. iii, p. 570.

bacterial invasion of the kidney. Acute hematogenous infections also have been much discussed in connection with the pyelitis of pregnancy, but any discussion of this disease would occupy more space than the limit of our paper permits. Louria[1] in a recent paper has given some consideration to this subject. Finally, we wish to quote in full the following remarks by Brewer.[2] He concludes from his experimental data: (1) That during the progress of any infectious disease a certain number of microörganisms find their way into the blood current, and that many of these organisms are excreted through the kidneys. If the number of these organisms is comparatively small, if their virulence is low, and if the kidneys are in a healthy condition, the transit of these organisms through the renal apparatus gives rise to no demonstrable lesions. If, on the other hand, the number of the organisms is large, if their virulence is high, or if one or both kidneys are diseased, lesions are produced which may at the outset cause an overwhelming and fatal toxemia, or may proceed more slowly to the development of any of the classical types of renal infection or suppuration; (2) that while the disease may be bilateral, in a large number of instances it is unilateral, and its unilateral character is due to the fact that the affected kidney has lost to some extent its normal resistance to infection by reason of trauma, abnormal mobility, previous disease, calculous irritation, anemia, passive hyperemia, complete, incomplete, or intermittent hydronephrosis; (3) that the presence in the body of a kidney damaged by trauma or disease to such an extent as to lower its normal resistance to infection is a distinct menace to the individual, in that it possesses a potential susceptibility toward even the mildest forms of blood infection; (4) that while it is possible to produce these lesions in animals by means of the B. coli, the Streptococcus pyogenes, the Staphylococcus pyogenes aureus, the B. typhosus, the pneumococcus, and the pyocaneus, in clinical cases only the first four of these organisms have been isolated.

[1] New York Medical Journal, 1911, vol. xciii, p. 1073.
[2] Jour. Amer. Med. Assoc., 1911, vol. lvii, p. 187.

Pathology. The microscopic appearance of the kidney varies with the stage of advancement of the morbid process. At first the kidney is swollen, tense, and engorged with blood; infarcts may be seen. Later, numerous yellow nodules appear beneath the capsule surrounded by a zone of intense congestion. Upon section these nodules are found to be the bases of wedge-shaped areas of suppuration in the cortex. Long, linear, yellow streaks may indicate suppuration in the tubules. The microscopic changes consist of a diffuse congestion and leukocytic infiltration with numerous foci of suppuration; there is often much hemorrhage beneath the capsule, and throughout the cortex triangular infarcts, hemorrhage, or suppuration may be present. Large bacterial masses looking like emboli are often seen. In the later stages the foci of suppuration coalesce and gross abscesses may be formed. The Malpighian tufts are congested and usually surrounded by a leukocytic infiltrate. The pelvis is congested and covered with exudate.

Symptoms. The clinical picture of hematogenous pyelonephritis varies considerably with the virulence of the process. Brewer describes three clinical types: (1) The severe type, in which the local symptoms are obscured by an intense toxemia and a fatal result occurs unless the infected kidney is removed; (2) the intermediary type, with severe initial symptoms but without grave toxemia. Renal abscess, perinephritic abscess, pyelonephritis, or pyonephrosis develops in neglected cases. The symptoms may simulate appendicitis, cholecystitis, or abscess of the liver, and if the renal disturbance is not marked, typhoid fever or pneumonia; (3) the mild type, with tenderness over the costovertebral angle as the only symptom, suggesting subacute appendicitis or cholecystitis. Brewer believes that this type accounts for certain irregular periods of temperature occurring during convalescence from some surgical conditions or infectious diseases. To these we will add a fourth type, that in which the chief and perhaps the only symptom is hematuria. We have referred to the observations of Gaudiani and would also add those

of Eshner[1], Billings,[2] Elliott,[3] and White[4] for additional support of the view that some of the so-called "essential hematurias" are due to bacterial activity.

The "severe type" of Brewer or the fulminating type of other writers is of sudden onset in a person apparently well. There is severe abdominal pain, tenderness, rigidity, nausea, and vomiting. While the pain at times is characteristic of renal disease, it generally suggests some intraperitoneal process on the side affected, usually of the stomach, gall-bladder, or appendix. A number of the cases reported in the literature were operated on for appendicitis, cholecystitis, or perforated duodenal ulcer before the true condition was recognized. The temperature rises sharply, even as high as 106° F., and there may be a chill or succession of chills. Septic symptoms develop and sometimes death may ensue before any distinctive renal or urinary signs are observed. As a rule, however, an indication of the true condition of affairs is given by the shifting of the pain to the flanks and the finding of a point of more or less severe tenderness at the costovertebral angle. It is rare that the enlarged kidney can be palpated owing to the rigidity present, although some resistance may be felt by bimanual examination. The urinary findings during the early hours are inconclusive and do not differ from those in any other severe septic infection, but later the urine becomes scanty and contains albumin, casts, blood, and pus cells, and the offending microörganism in pure or mixed culture. There is usually a high degree of leukocytosis, and headache, muscular twitchings, convulsions, or delirium may be present. The fatal termination is due to a combination of sepsis and uremia.

The second type, the *forme aigue* of Albarran, resembles the foregoing except that the intense septic symptoms are absent. More time is given in which to make a diagnosis, and the costovertebral tenderness and the urinary findings can be supplemented by ureteral catheterization. The bladder will be seen to be intensely inflamed about the ureteral orifice from which

[1] Amer. Jour. Med. Sci., April, 1903.
[2] Ibid., 1910, p. 625.
[3] International Clinics, vol. iv, sixth series, p. 122.
[4] Quarterly Journal of Medicine, 1911.

pus may be seen exuding, and the lips of the orifice will be edematous and pouting and often ulcerated. The cystoscopic examination should not be made during the acute attack unless nephrectomy is contemplated. An *x*-ray examination should be made to determine the presence or absence of calculus.

The mild type which Brewer also terms "idiopathic pyelitis" has but few symptoms except moderate pain (backache) and costovertebral tenderness. A trace of albumin and a few casts, blood cells, and pus cells will be found in the urine. Some of these cases have no doubt been operated upon for subacute appendicitis or cholecystitis, but on the other hand they may account for "certain irregular periods of temperature occurring during convalescence from some surgical condition or infectious disease" (Brewer). The fourth type is characterized by persistent unilateral hematuria which cystoscopy shows to come from the kidney, and the cause of which is revealed only at operation.

Treatment. The acute fulminating form should be operated upon as soon as the diagnosis can be made, with the intention of performing nephrectomy unless nephrotomy with drainage will suffice. In 1911 Brewer reported that he had encountered 14 cases of the severe type: two were untreated and in four nephrotomy and drainage were done; they all died. Eight were treated by early nephrectomy and all recovered. The general statistics on the subject are not yet susceptible of analysis. It is quite true that the fulminating type sometimes subsides without operation, but just as in acute appendicitis we never know which cases will recover and which will not. Of greater importance is the question of whether to do a nephrectomy or a nephrotomy. Brewer and Cobb have obtained splendid results from nephrectomy, but there is much to be said for the less radical procedure, chiefly because of the uncertainty as to the condition or even the presence of the other kidney, since there is rarely time for a cystoscopic examination. The objections that have been urged against nephrotomy are the inability to drain the whole organ by splitting it and the subsequent uselessness or even the menace of a kidney riddled with abscesses. Cases are on record in which

the symptoms recurred after nephrotomy, necessitating a subsequent nephrectomy. In the subacute types, more time is given for investigation of the other kidney and the diseased organ can be more successfully treated. In the case herein reported we adopted a conservative plan of treatment for nearly three weeks, and when forced to operate should have been content with a nephrotomy if the friable, disorganized state of the kidney had permitted it. Of course, in the mild cases operation is not to be thought of; rest in bed, a milk diet, urinary antiseptics, purgatives, and plenty of water will usually bring about a cure. In refractory cases bacterins may be used and in persistent pyelitis it may be necessary to irrigate the renal pelvis through a ureteral catheter with a solution of one of the silver salts or with a 2 per cent. solution of aluminum acetate. If still resistant, it is advisable to operate, split the capsule and drain the renal pelvis. In all such cases the existence of pressure on the ureter or of movable kidney should be investigated.

DISCUSSION

Dr. B. A. Thomas: I am reminded of a case which I saw in Dr. Frazier's service, of acute hematogenous unilateral nephritis, with practically the identical symptomatology as presented in this case. There was an acute onset characterized by sharp pain in the region of the right kidney, which after the onset was marked by the continuation of the pain of a dull character. When the patient was seen two or three days after the onset, she had tenderness in the right hypochondriac region and tenderness on palpation in the right costolumbar angle. An interesting feature was that this particular patient had had for a number of weeks previous to the onset a chronic bronchitis, and it was my opportunity on this occasion to catheterize the ureters and to carry out a functional kidney test. The indigocarmine test was used, and the dye was eliminated from both kidneys in seven minutes, from which the inference was drawn that the kidneys were functionally sufficient. Nevertheless, in view of the pain, tenderness, etc., we believed that something was wrong, and catheterization of both sides was done and the bacillus of Friedländer was found to be the infecting organism. This

was of particular interest in view of the previous condition of chronic bronchitis.

With respect to the classification of suppurative nephritis it has always seemed desirable and more advantageous to classify the disease from the etiological standpoint. Some, however, claim that it is necessary for a mixed infection to exist in order to produce the suppurating condition. This is probably true when the colon bacillus is the primary invading organism, but certainly such is not the case when the infective bacterium is of many another species. It has, therefore, become common to classify suppurative inflammation of the kidney on the pathologico-anatomical basis, and we have thus classified it under three groups:

1. The formation of a single large focus which may become confluent with its neighbor, giving rise in the parenchyma of the kidney to what may be called abscess of the kidney.

2. We may have, on the other hand, numerous small foci giving rise to miliary lesions, which we describe as suppurative nephritis. The lesions may or may not, later, rupture into the pelvis of the organ.

3. In case the disease ascends or is urogenous in origin, it first gives rise to an inflammation of the kidney pelvis, and may then spread, and secondarily infect the parenchyma of the organ, producing the well-known condition of suppurative pyelonephritis.

One other point to which I would like to call attention with respect to the unilateral character of these infections of the kidneys, is that probably in a great number of cases the unilateral origin may be due to lowered resistance on that particular side incident to traumatism—a *locus minoris resistentiæ*, if you please. This may not necessarily be due to a fall or blow. It may be the result of a contortion of the body, such as overflexion or extension.

With respect to the avenue or source of infection, Küster claimed that in the majority of cases it is urogenous. We know that in a small percentage it is lymphogenous. The present belief is unquestionably to the effect that the large majority of cases are hematogenous in origin. Particularly is this so if we may exclude urethral obstruction as an etiological factor giving rise to interference with the urinary efflux.

Dr. John B. Roberts: Dr. Riesman's scientific spirit and report have recalled to mind a case seen about five years ago with Dr. J. Dutton Steele, which strikes me as being of the same sort. The patient was a woman with swelling, pain, etc., in the left portion of the abdomen and loin, with changes in the urine. She was examined carefully, and the conclusion reached that there was suppurative inflammation of that one kidney. We called in Dr. Willard, who agreed with our decision. The woman was sent to the Presbyterian Hospital, and Dr. Willard skilfully cut into the kidney through the loin, and emptied an abscess. He put in a drain for a time, and the patient recovered without a fistula,

and lived for a number of years. She only recently has died. In my ignorance, I at that time had the opinion, held by a good many, that most of these cases were ascending nephritis of urological causation. In this case the husband had had previously a prostatectomy for enlarged prostate, which had left either a vesicorectal or urethrorectal fistule. I jumped to the conclusion that this renal abscess was due to an ascending nephritis from cystitis in the husband. The symptoms were so similar to those cases of Dr. Brewer, of New York, and that of Dr. Riesman, that it is probable that the case was one of similar etiology. If it is true that the majority is hematogenous rather than urogenous we must see them quite often. Although I have not Dr. Steele's records, it seems to me the case is similar to those described by Dr. Brewer and that Dr. Riesman has brought forward a case of similar nature.

Dr. James Tyson: I have been much interested in Dr. Riesman's report of this case and his exposition of the subject, as well as the conclusions which he has drawn from it. I have never myself seen a case of acute unilateral hematogenous nephritis produced by the ordinary causes of a nephritis. On the other hand, it is almost never that you see a fairly symmetrical involvement of both kidneys. It is not unusual to see one organ as large and larger than normal, while the other is much reduced in size. This implies a greater vulnerability on the part of the one kidney, which may explain a part of the unilateral cases of various kinds that are met. I think a certain proportion of the cases may be due to adjacent foci of pathogenic organisms like the colon bacillus, the streptococcus, and probably the tubercle bacillus. In the case of multiple superficial abscess of the kidney as illustrated by the first of the pictures thrown on the screen, I think an embolic origin is more common, although I think, too, this sort of kidney is rarely unilateral. In the Wood Museum of the University of Pennsylvania there is a wax reproduction of the natural appearance of such a kidney as I should suppose this one represented. Thus we may have a certain number of cases which are embolic in their origin. The remaining cases, it seems to me, can be explained by reason of a greater virulence of the infecting agent or by a greater vulnerability of one of the kidneys. I believe that the experience of those working in the lines of the cystoscopic examination and catheterization of the ureters will show a larger proportion of the unilateral cases than have been recognized by the usual methods of investigation.

The unilateral hemorrhagic cases are most difficult of explanation. The most that can be said is that such a kidney has been invaded to the exclusion of its companion by a virulent toxic substance which acts like the causes of purpura and other hemorrhagic conditions of more superficial situations which we see in different parts of the body.

APPENDIX

ANNUAL REPORT OF THE LIBRARY COMMITTEE FOR 1912

MR. PRESIDENT: In accordance with the ordinances and By-Laws of the College, I herewith submit the following report of the Library Committee for the year 1912:

Total number of volumes in the Library, including the bound volumes and 8600 unbound "Reports" and "Transactions"	97,803
Number of unbound "Theses" and "Dissertations"	10,123
Number of unbound pamphlets	75,125

Included in the above total there are 3041 volumes known as "reserves," consisting of second copies of some of the more important periodical publications, and 2926 volumes more or less incomplete.

In addition, the duplicates at this date number 3926 volumes.

The following table shows the number of volumes in the various divisions of the Library:

	Bound.	Incomplete and unbound.	Total.
General Library	69,042	2863	71,905
Lewis Library	13,543	44	13,587
On permanent deposit:			
S. D. Gross Library	3,491	3	3,494
Library of the Obstetrical Society of Philadelphia	217	0	217
			89,203

The Library has received from all sources, during the past year, 4317 volumes, 7345 pamphlets, and 32,194 numbers of various periodicals. Of these 285 were received in exchange.

Accessions (including 106 volumes of "reserves"):

General Library	2840
Lewis Library	14
S. D. Gross Library	46
	2900

The individual "donors" for the year ending November 1, 1912, number 553; this represents 1049 distinct presentations. Every gift, whether it be a single reprint or a large number of volumes, is duly acknowledged and properly recorded.

The following list shows the donations of twenty-five volumes or more, the number of volumes presented by the various publishing houses, and the gifts of the Philadelphia Pediatric Society:

	Volumes.
Dr. E. L. Duer	339
Dr. De Forest P. Willard	244
Dr. S. W. Morton	132
Dr. J. B. Roberts	107
Mr. William Janney	94
Dr. H. A. Hare	73
Dr. W. W. Keen	61
Dr. W. G. Spiller	48
Mrs. J. B. Walker	48
Dr. F. R. Packard	43
Dr. A. A. Snyder	41
Dr. G. E. de Schweinitz	38
Dr. G. M. Marshall	34
Dr. E. H. Goodman	32
Dr. H. M. Fisher	30
Dr. S. Weir Mitchell	29
From the publishing houses of:	
D. Appleton & Company	5
P. Blakiston's Son & Company	39
F. A. Davis Company	11
Lea & Febiger	6
J. B. Lippincott Company	22
W. B. Saunders Company	44
William Wood & Company	9
From the Philadelphia Pediatric Society:	3

The Library is indebted for large gifts of pamphlets and unbound periodicals to the following gentlemen:

Dr. J. M. Anders
Dr. A. P. C. Ashhurst
Dr. E. J. G. Beardsley
Dr. H. W. Cattell
Dr. Burton Chance
Dr. S. Solis Cohen
Dr. E. P. Davis
Dr. G. G. Davis
Dr. E. L. Duer
Dr. C. W. Dulles
Dr. A. A. Eshner
Dr. T. H. Fenton
Dr. D. W. Fetterolf
Dr. John V. Fisher
Dr. C. H. Frazier
Dr. J. S. Gibb
Dr. J. H. Girvin
Dr. E. H. Goodman
Dr. H. A. Hare
Dr. C. J. Hatfield
Dr. F. P. Henry
Dr. A. B. Hirsh
Dr. L. W. Johnson
Dr. Bayard Kane
Dr. W. W. Keen
Dr. R. G. Le Conte
Dr. G. M. Marshall
Dr. Edward Martin
Dr. John K. Mitchell
Dr. S. Weir Mitchell
Dr. C. C. Norris
Dr. C. A. O'Reilly
Dr. F. R. Packard
Dr. J. B. Roberts
Dr. P. J. Sartain
Dr. T. B. Schneidemann
Dr. B. Schwartz
Dr. G. E. de Schweinitz
Dr. P. N. K. Schwenk
Dr. J. J. Taylor
Dr. James Thorington
Dr. E. Q. Thornton
Dr. W. H. Trueman
Dr. H. R. Wharton
Dr. J. William White
Dr. J. C. Wilson
Dr. A. C. Wood
Dr. H. C. Wood

Also to Messrs. P. Blakiston's Son & Co., J. B. Lippincott Company, William B. Saunders Company, and John Wyeth & Brother.

724 new publications were added to the Library during the past year; 41 of these works were written or edited by Fellows of the College.

30 volumes were presented by the following authors or editors:

Dr. Paul Bar (Paris)
Dr. George E. Brewer
Dr. E. de Cyon (Paris)
Dr. T. Diller
Dr. Seneca Egbert
Dr. Martin H. Fisher
Dr. Hobart A. Hare (editor)
Dr. Edward Jackson
Dr. Otto Juettner
Dr. Howard A. Kelly
Dr. H. R. M. Landis (editor)
Dr. F. La Torre (Paris)
Dr. J. Ewing Mears
Sir William Osler, M.D., Bart.
Dr. William Pepper
Dr. L. S. Pilcher
Dr. C. S. Potts
Dr. John B. Roberts
Sir James Sawyer, M.D.
Dr. James Thorington
Dr. James J. Walsh
Dr. T. H. Weisenburg
Dr. Royal Whitman
Dr. Casey A. Wood
Dr. T. A. Woodruff

18 volumes were sent by the publishers at the request of the following authors or editors:

Dr. B. K. Ashford
Dr. A. P. Brubaker
Dr. H. W. Cattell (editor)
Dr. R. M. Goepp
Dr. G. M. Gould
Dr. H. F. Hansell
Dr. J. C. Heisler
Dr. P. G. Igaravidez
Dr. Howard A. Kelly
Dr. E. E. Montgomery
Dr. John H. Musser
Dr. Walter L. Pyle
Dr. Wendell Reber
Dr. James Thorington

Summary of the "Funds":

	Volumes purchased.	Cost.	Volumes bound.	Cost.
Henrietta Rush Fales Baker Fund	67	$213.54	15	$12.55
Luther S. Bent Fund	22	48.78	5	3.55
William T. Carter Fund	61	258.67	15	14.65
Girardus Clarkson Fund	12	41.64	5	2.10
Francis X. Dercum Fund	66	250.39	10	4.95
John D. Griscom Fund	80	277.08	21	14.95
William F. Jenks Fund	124	515.08	15	9.00
Oliver A. Judson Fund	16	63.78	3	1.45
Wm. V. and John M. Keating Fund	14	55.14	3	2.15
William W. Keen Fund	71	264.89	12	7.75
Library Endowment Fund	156	660.96		
Charles K. Mills Fund	1	1.32		
Weir Mitchell Fund	59	240.46		
John H. Musser Fund	7	24.56		
Elizabeth K. Newcomet Fund	18	34.72	4	2.15
William F. Norris Fund	42	167.08	12	10.45
Philadelphia Medical Society Fund	4	16.40		
Lewis Rodman Fund	50	282.06	4	10.50
Douglas Stockton Warren Fund	7	24.30		
John F. Weightman Fund	19	52.67	8	3.50
Caspar Wistar Fund	43	215.16	5	3.00
Total	939	$3708.68	137	$102.70

Special Accounts:

	Volumes purchased.	Cost.	Volumes bound.	Cost.
Fund for completing files of journals	119	$330.64		
Fund for rare and valuable books	24	299.62		
Journal Association	20	139.50		
New Book Fund	40	137.02		
J. Ewing Mears Account	26	91.63	6	$3.05
S. D. Gross Library Account	41	228.72	4	5.30
Total	270	$1227.13	10	$8.35

George B. Wood Fund for Library supplies, stationery, etc. Expended $272.89.

There were 1547 volumes bound during the past year. This includes all new books and current periodicals, and 370 volumes of pamphlets and reports.

	1912.	1911.	Increase.
Number of visitors to the Library	7329	6157	1172
Fellows of the College	3029	2955	74

The Library has been kept open two evenings each week, and on the six minor legal holidays, for the same hours and period of time as during the preceding year:

	1912. (80 evenings)	1911. (77 evenings)	Increase.
Visitors, evening	545	410	135
Fellows of the College	199	185	14
Visitors, legal holidays	133	100	33
Fellows of the College	40	34	6

The above figures are included in the total number of visitors for the year.

	1912.	1911.	Increase.
Number of books consulted in the Library	17,146	16,003	1143

The number of books reported as "consulted in the Library" includes only those supplied by the Librarian or his assistants. Readers have access to the bound volumes kept on the shelves

in the "Reading Room," and the Fellows of the College have access to the "book stacks." There are, therefore, a great many volumes consulted of which no accurate record can be kept.

	1912.	1911.	Increase.
Number of books taken out	3758	3643	115

	Works.	Volumes.	Cards written.
Cataloguing	985	1165	10,123

All the books added to the shelves during the past year have been catalogued, and each volume accessioned and shelf-listed. Of the unbound pamphlets and reprints received during the year 5384 have been retained and subject-headed.

2455 new shelf-list cards have been typewritten. This completes the typewritten card shelf-list of the General Library.

Revision of catalogue: Number of cards revised, typewritten, examined, and alphabeted for the year ending November 1, 1912, 20,000.

The revision of the catalogue has been completed as far as the second series of the "Index-Catalogue" has been issued—"Stysanus"—and the work will be finished as rapidly as the remaining volumes of the alphabet appear. The shelf-list cards of the General Library, which are now complete to date, are a part of the revision; this list, together with the lists of the Lewis and Gross libraries, were formerly in book form. Cards for the Lewis and Gross libraries are now being typewritten, as time permits in connection with the regular duties. The shelf-list is a facsimile, on cards, of the arrangement of the books upon the shelves, and is used for stocktaking, and for tracing missing books when only the class and number are known.

Under the head of "revision" mention can properly be made of the change in the arrangement of unbound pamphlets and reprints. The Library Committee, September 23, 1912, approved the recommendation of the Librarian, and ordered that "the unbound pamphlets and reprints in the Library of the College

of Physicians be arranged by subjects, and alphabetically by author under the subject, following the plan adopted in the 'Index-Catalogue of the Library of the Surgeon-General's Office;' and, further, that the proper subject-heading be written upon each pamphlet or reprint." Formerly the unbound pamphlets and reprints were arranged by classes, the same as the books upon the shelves. This has not proved satisfactory. Books, from the nature of their contents, in most cases, can only be arranged by classes and their subdivisions; unbound reprints, on the other hand, are almost invariably upon a single subject, and therefore each pamphlet can have its correct subject-heading written upon it, and arranged upon the shelves alphabetically by subject, as well as by author, under the subject. This arrangement forms a subject catalogue of pamphlets upon the shelves, in the same manner as cards written for the subjects and placed in the catalogue trays. When the pamphlets accumulate, from twenty to fifty, according to their size, on one subject or division of a subject, they can be bound in a volume and placed with the books under the proper class. One card under the subject or subdivision of the subject, as the case may be, placed in the catalogue, covers the twenty or fifty pamphlets in the volume, and makes available to the reader or student a collection of original articles on a particular subject which could be obtained otherwise only by referring to numerous periodicals, after the references had been obtained. To catalogue each pamphlet would not only be a waste of time, but would fill up the catalogue with useless cards; and economy of space is of great importance especially, as in the present instance, where the room allotted for catalogue trays is limited.

This additional work has been added to the duties of the Catalogue department, at present, principally under the head of "revision." Up to the present time 200 volumes, representing 4036 pamphlets and reprints, have been bound by subjects, and 5384 unbound pamphlets and reprints have been subject-headed and arranged alphabetically by subject, and by author under the subject, upon the shelves.

The current periodical publications, including "Transactions" and "Reports," received as issued, at this time are obtained through the following sources:

	American.	Foreign.
Henrietta Rush Fales Baker Fund	0	30
William T. Carter Fund	2	24
Francis X. Dercum Fund	0	12
John D. Griscom Fund	4	39
Samuel D. Gross Library Account	0	3
William F. Jenks Fund	3	38
Journal Association	1	11
Oliver A. Judson Fund	0	6
William V. and John M. Keating Fund	0	7
William W. Keen Fund	3	24
Library Endowment Fund	4	113
J. Ewing Mears Account	0	1
Weir Mitchell Fund	1	33
Elizabeth K. Newcomet Fund	0	1
William F. Norris Fund	1	10
Philadelphia Medical Society Fund	0	6
Lewis Rodman Fund	0	14
John F. Weightman Fund	0	4
Caspar Wistar Fund	4	19
By Purchase (General Account)	14	176
In Exchange	88	82
Editors	120	21
Publishers	41	1
Total	286	675
In addition, current numbers of periodicals are received at stated intervals through the courtesy of the editors and editorial staff of the following journals: American Journal of the Medical Sciences, Medical Council, Therapeutic Gazette.	188	105
	474	780

Total number of periodical publications received at this date, 1254.

Increase in subscriptions for the year: American, 23; foreign, 109. Total, 132.

We exchange publications with the following schools of medicine:

University of	Basel.	University of	Kiel.
"	Berlin.	"	Königsberg.
"	Bern.	"	Lausanne.
"	Bonn.	"	Leiden.
"	Breslau.	"	Leipzig.
"	Erlangen.	"	Lund.
"	Geneva.	"	Marburg.
"	Giessen.	"	Rostock.
"	Göttingen.	"	Strassburg.
"	Greifswald.	"	Upsala.
"	Halle.	"	Würzburg.
"	Heidelberg.	"	Zurich.

Faculty of Medicine of Bruxelles.
" " Paris.
" " Toulouse.
" " Yucatán.

The University of Lund (Universitets-Biblioteket, Lund) has been added to the list during the past year, as has also the Faculty of Medicine of Yucatán (Escuela de Medicina, Cirugia y Farmacia de Yucatán).

1206 Dissertations have been added to the Library during the past year.

We have received for the current year ending November 1, 1912, $206.89 in cash from the sale of duplicates. 125 volumes and 8030 numbers of journals were received in exchange, arranged on a cash basis, from two New York dealers.

During the year we have distributed duplicate books and journals on exchange account to the following:

Boston Medical Library.
Lane Medical Library, San Francisco.
United States Naval Medical School Library.
University of Michigan.
University of Missouri.
University of Pennsylvania.

Returns have been received from the Boston Medical Library and the United States Naval Medical School Library.

With the aid of exchanges, and by purchase, from funds appropriated for the purpose by the Library Committee, we have, since November 1, 1911, completed our files of the following journals:

Archiv für Anatomie und Physiologie.
Archiv für Anatomie, Physiologie und wissenschaftliche Medicin.
Archivio italiano di ginecologia.
Bulletin de la Société française d'histoire de la médecine.
Le Concours médical.
La Ginecologia.
Revista di clinica pediatrica.
Revue clinique d'andrologie et de gynécologie.
Revue pratique des maladies des organes génito-urinaires.
Schweizerische Blätter für Gesundheitspflege.
Zeitschrift für Morphologie und Anthropologie.
Zentralblatt für chirurgische und mechanische Orthopädie.

Amount of fines collected from November 1, 1911, to November 1, 1912, $19.65.

The following is a list of the rare medical books and works of special interest received during the past year.

Incunabula

(Total number of incunabula at this date, 170)

Barzizius, Christoforus. Introductorium ad opus practicum medicinæ cum commentariis in ix Almansoriis. Papiae, Antonius de Carchano, 1494. [Hain no. 2666.] This edition is extremely rare. Only three copies mentioned by Pellechet. No copy either in the British Museum or in the Library of the Surgeon-General's Office. (Presented by Dr. George E. de Schweinitz.)

Burley, Walter. De vita et moribus philosophorum et poetarum. [Parisiis, Geo. Mittelhus, 1496.] [Copinger no. 1389.] (Fund for rare books.)

Culmacher, Philipp von Eger. Regimen wider die Pestilenz. [circa 1480.] [Hain no. 5848.] Not in the "Index-Catalogue" or in any bibliographical work which we have been able to consult; and, from inquiries we have made, it seems probable that it is the only copy in this country. (Presented by Dr. George E. de Schweinitz.)

Diogenes Cynicus. Epistolae. Diogenes epistolae interprete Francisco Aretino, Bruti et Hippocratis epistolae per Rainucium traductae. Florentiae, Antonium Francisci Venetum, 1487. [Hain-Copinger no. 6193.] Very rare. (Fund for rare books.)

Dondus Paduanus, Jacobus. Aggregator Paduanus de medicinis simplicibus. [Argentorati, Rusch, circa 1470.] [Hain no. 6395.] Dr. Garrison, of the Library of the Surgeon-General's Office, says of this work, "the earliest known of medical incunabula." This is a perfect copy in excellent condition. (Presented by Dr. George E. de Schweinitz.

Gerson, Johannes. De cognitione castitatis et pollutionibus diurnis, etc. [Coloniae, Ulrich Zell, circa 1470.] [Hain no. 7691.] (Presented by Dr. Richard H. Harte.)

Gerson, Johannes. De pollutione nocturna, an impediat celebrantem vel non. [Coloniae, Ulrich Zell, circa 1472–73.] [Hain no. 7695.] This is a rare edition, in a wonderfully good state of preservation, from the first press at Cologne. (Presented by Dr. S. Weir Mitchell.)

Johannes Peachamus. Archbishop of Canterbury. Prospectiva communis. [Mediolani, Petrus Cornenus, circa 1480.] [Hain no. 9425.] Deals largely with the eye and optics. Very rare. (Fund for rare books.)

Paulus Venetus, Nicolettus. Expositio librorum naturalium Aristotelis. Venetiis, Johannis de Colonia, 1476. [Hain no. 12515.] (Fund for rare books.)

Savonarola, Hieronymus. Ricetto contra morbo spirituale. [Florentiae, Bartholomaeus de Libris, 1497.] [Hain no. 14371.] Very rare. (Fund for rare books).

Works of Special Interest

Aldrovandus, Ulysses. De reliquis animalibus exanguibus libri quatuor. Bononiae, Bellagambam, 1606. De quadrupedibus solipedibus volumen integrum. Bononiae, Thebaldinum, 1639. Serpentium et draconum historiae, libri duo. Bononiae, Ferronii, 1640. Quadrupedum omnium bisulcorum historia. Bononiae, Ferronii, 1642. De quadrupedibus digitatis viviparis et oviparis. Bononiae, Tebaldinum, 1645. All the writings of Aldrovandus are considered rare, and these five volumes are exceptionally fine copies, in perfect condition, which greatly enhances their value. (Presented by Sir William Osler, M.D., Bart.)

Alexander Aphrodiseus et Michael Epheseus. Simplicii commentaria in tres libros Aristotelis de anima. Venetiis, Aldi Manutii, 1527. (Fund for rare books.)

Alghisi, Tommaso. Litotomia, ovvero del cavar la pietra. Firenze, Manni, 1707. (Presented in the name of Dr. Alfred Stillé.)

Bacci, Andrea. Del Tevere. Libri tre. Venetia, 1576. (Fund for rare books.)

Bauderon, Bricius. Paraphrase sur la pharmacopée. 2d ed. Lyon Servain, 1596. This is a rare edition of what, in its time, must have been a popular book. The Index-Catalogue does not contain this edition, and no mention is made of it in Brunet. There is a book-plate on the inside of the first fly-leaf which shows that this copy was once in the possession of the "Yemeniz Library"; and a leather label on the inside of the cover, that it was later in the Robert Hoe Library. This book was bound by Trautz-Bauzonnet. (Presented by Dr. George E. de Schweinitz.)

Beroaldus, Philippus. Opusculum de terraemotu et pestilentia. Bononiae, Benedictus, 1515. (Fund for rare books.)

Bibliotheca anatomica. 2 vols. Genevae, Chouët, 1685. (Presented by Mrs. Henry C. Chapman.)

Bottarelli, Giovanni. De bagni di San Casciano. Firenze, Vangelissi, [1688]. (Fund for rare books.)

Brown, William. To the medical officers of the general and flying hospitals in the army of the United States. Philadelphia, 1780. [Broadside.] (Presented by Dr. William Pepper.)

Browne, John. Compleat treatise of the muscles. [London], Newcombe, 1681. This is a fine copy of a rare work printed in English, and noted for its illustrations. (Presented by Dr. George E. de Schweinitz.)

Carnarius Gandensis, Joannes. De podagrae. Patavii, 1553. (Fund for rare books.)

de l'Epée, Charles Michel l'Abbé. Institution des sourds et muets. Paris, Nyon, 1776. (Fund for rare books.)

a Fonseca, Rodericus. De calculorum remediis. Romae, Ruffinellus, 1586. (Fund for rare books.)

Galen, Claudius. Exhortatio ad bonas arteis. Basileae, Froben, 1526. (Presented in the name of Dr. Alfred Stillé.)

Galen, Claudius. Libri duo.*** Astrologia ad aphrodisium. De urinis. Venetiis, [Patavinus] 1535. (Fund for rare books.)

Garcia, Mathias. Disputationes medicinae selectae. Lugduni Bourgeat, 1677. This work must be quite rare. Not in Brunet, or in the other bibliographical works consulted. Not in the Index-Catalogue of the Surgeon-General's Office. (Presented by Dr. F. P. Henry, in the name of Dr. Herbert Norris.)

Glauber, Joh. Rudolph. De Elia artista. Amsterdam, Waesberge, 1668. (Fund for rare books.)

Glauber, Joh. Rudolph. De igne secreto philosophorum. Amsterdam, Jansson, 1669. (Fund for rare books.)

Glauber, Joh. Rudolph. Glauberus concentratus. Amsterdam, Waesberge, 1668. (Fund for rare books.)

de Gradi, Joannes Matthaeus Ferrarius, Practica. Venetiae, Scoti, 1520. (Fund for rare books.)

Harvey, William. Exercitationes de motu cordis et sanguinis. Edinburgi, Carfrae, 1824. (Presented by Dr. A. P. Brubaker.)

Hippocrates. Chymicus; seu, Chimicae Hyppocraticae. Coloniae, Vanden Dyck, 1690. Copies of this little book are very rare. Not mentioned by Poggendorff, Brunet, Graesse and other bibliographers. Prof. John Ferguson, in his Bibliotheca chimica, vol. 1, p. 118, states that he has never seen a copy. Not in the Library of the Surgeon-General's Office. (Fund for rare books.)

Lemnius [Ziriczaeus], Laevinus. Secret miracles of nature. London, Streater, 1658. (Presented by Dr. Bayard Kane.)

Redi, Francesco. Osservazioni intorno agli animali viventi. Firenze, Matini, 1684. (Presented by Sir William Osler, M.D., Bart.)

Sanctorius, Sanctorius. De statica medicina aphorismorum. Londini, Bowyer, 1716. (Presented by Dr. George W. Norris.)

Savonarola, Giovanni Michele. Libreto de tutte le cose che se manzano comunamente. Venetia, de Luere, 1508. (Fund for rare books.)

Sophia, Marsilius de Sancta. Tractatus celeberrimus de febribus. Lugduni, Trot, 1507. (Fund for rare books.)

Tagaultius, Johannes. De chirurgica institutione libri quinque. Lugduni, Rovillium, 1560. (Fund for rare books.)

Thornton, Robert John. Preuves de l'efficacité de la vaccine. Paris, Chomel, 1808. (Presented by Dr. George E. de Schweinitz.)

Valverde, Joan. De animi et corporis sanitate tuenda libellus. Venetiis, Lilius, 1553. (Fund for rare books.)

Victorius Faventinus [de Victoriis], Leonellus. Practica medicinalis. Venetiis, Constantinum, 1553. (Fund for rare books.)

Zacchias, Paulus. Quaestionum medico-legalium. Francofurti a. M., Bencard, 1688. (Presented by Mrs. Henry Chapman.)

Manuscripts

de Benneville, George. Medicina Pensylvania. [Philadelphia, circa 1770.] (German and English text.) (Presented by L. G. Myers, Esq.)

Coates, Samuel. Autograph letter to his kinsman, Joseph Paschall, September 25, 1793. Philadelphia, 1793. Written during the yellow fever epidemic and interesting on account of its reference to Rush. (Presented by Dr. William Pepper.)

Massa de Argenta, Baptista. De fructibus vescendis. [Ferrariae, 1471.] The printed book is one of the earliest dated incunabula, and extremely rare. Maggs Brothers, of London, from whom the present book was obtained, made the following statement: "probably the original

manuscript from which the book was printed." (Presented by Dr. George E. de Schweinitz.)

Redman, John. Autograph letter to the Rev. Ashbell Green, Princeton, September 14, 1798. [Philadelphia], 1798. Written and signed by the first President of the College. (Presented by Dr. William Pepper.)

Shippen, William, Sr. Autograph letter to George Washington, December 10, 1783. Philadelphia, 1783. Written by Dr. William Shippen, Sr., father of the second President of the College. This is a letter of congratulation, written in the third person. Endorsed in Washington's handwriting. (Presented by Dr. William Pepper.)

Other Interesting Accessions

Aesculapius. A very handsome print of E. J. Poynter's painting entitled: A Visit to Aesculapius. (Presented by Dr. S. Weir Mitchell.)

Rabelais, Francois. Photograph of Rabelais' portrait in the Library of the University of Geneva. Dr. Charles G. Cumston, of Boston, from whom it was purchased, says: "This is, I believe, the only authentic likeness of the great physician."

Royal College of Physicians of London. A colored print of a meeting of the Fellows of the College of Physicians (London). Published May 1, 1808. Rowlandson & Pugin, Delt & Sculpt. Bluck Aquat. Colored prints of this description are rare and quite difficult to obtain. (Presented by Dr. Richard H. Harte.)

Badge with bronze medal attached, worn by members of the Ninth International Otological Congress, August 12 to 17, 1912. (Presented by Dr. B. Alexander Randall.)

Massive oak desk. (Presented by Dr. J. Norman Henry.)

Shoe buckle worn by Benjamin Rush. (Presented by his great-granddaughter, Elizabeth Rush Porter.)

In many respects this year's report is extremely gratifying; the figures show a marked increase in the number of readers (1172), as well as an increase in the number of volumes consulted, and the number of books taken out; also that the use of the Library by the Fellows of the College has increased. The evening hours show an increase in the total number of visitors recorded, and the average attendance of the Fellows of the College is slightly higher than for the past year. Last year special note was made of the increased use of the "Study Rooms," the average number of volumes called for and retained in these rooms being about 150; the records kept for this year give an average of 142 volumes.

The decrease in the number of unbound "Reports" and "Transactions," as well as unbound pamphlets, is accounted for by the fact that 5091 of these have been bound in 370 volumes and placed in their proper class upon the shelves.

The Library Committee endeavor each year to make such appropriation, as their funds will permit, for repairing and rebinding books found in a damaged condition, either from age or use; as well as for the binding, for their preservation, of such publications issued in the form of pamphlets, as can be properly grouped in volumes.

Through the generosity of Dr. Francis R. Packard, locked steel bookcases with plate wire glass doors, finished in mahogany to match the other furnishings, have been erected in the "Packard Room" to accommodate the valuable collection of manuscripts belonging to the College.

The Library Committee have had bookcases of the same style and construction built in Study Room No. 1, to accommodate the collection of books known as the "Lewis Curios," as well as the rare and valuable books, other than incunabula, belonging to the Library, that it is thought desirable to remove from the stacks and keep in locked cases. It is earnestly hoped that some provision will be made to furnish this room appropriately, and that the College will authorize that it be named the "Samuel Lewis Room," in memory of Dr. Samuel Lewis, one of the great benefactors of the Library. When Dr. Lewis made his first gift of books in 1864, the following Resolution was offered by Dr. Alfred Stillé, at that time Chairman of the Library Committee, at a stated meeting of the College, March 2, 1864: "*Resolved*, That the thanks of the College are hereby presented to Dr. Samuel Lewis for his magnificent gift of more than 2500 volumes of medical books, and that they shall be preserved as a separate collection under the name of the Lewis Library." Dr. Lewis was well aware, long before his death (1890), that the modern arrangement of books by classes and their storage in book-stacks, made it undesirable to receive or hold separate collections, and acquiesced in the fact that when the necessity arose the books of the Lewis

Library should be arranged in their proper classes with the other books belonging to the College.

It may be possible at some time in the future to arrange a room with the works of famous physicians, illustrating the discoveries and advancement in the science of medicine, from the inception of printing; each book to have a card inserted with explanatory note, showing why the author's writings were given a place in the collection. This would make an extremely interesting exhibit.

Three "Library Funds" have been received by the College during the past year: the "John H. Musser Library Fund," $500 (it was Dr. Musser's intention at the time of the gift to make this a fund of five or ten thousand dollars, and had he lived this would undoubtedly have been carried out); the "Douglas Stockton Warren Memorial Fund," $3000, and the "J. Ewing Mears Library Fund," $2000. In his letter to the College Dr. Mears states: "It is my expectation to add, within the next year, three thousand dollars ($3000) to complete the sum of five thousand dollars ($5000) which I have fixed as the amount of the Fund." The Library Committee has also received notification from the Secretary that the College, at its stated meeting November 7, "accepted a donation from Dr. John B. Roberts of five hundred dollars ($500) which he has given as a nucleus of a Catalogue Endowment Fund."

Respectfully submitted,

William J. Taylor,
Chairman.

REPORT OF THE COMMITTEE ON THE MÜTTER MUSEUM, 1912

The Chairman of the Mütter Committee has to report that the collections of the Museum have been frequently studied during the last year by Fellows of the College who have availed themselves of the material for illustrations to articles on various subjects.

There have been many contributions, among which may be especially mentioned:

1. Amputation of leg. Presented by Dr. M. B. Biggs.
2. Surgical case. Presented by Dr. J. M. Barton.
3. Small pocket medicine case. Presented by Dr. Sidney L. Olsho.
4. Case of instruments. Presented by Dr. James Darrach.
5. Preparation of penis. Presented by Dr. William M. Welch.
6. Thermometers and cupping instrument. Presented by Dr. Charles Baum.
7. Case of instruments. Presented by Dr. E. L. Duer.
8. An etruscan necklace. Presented by Dr. Thomas Biddle.
9. Fetus (seven months). Presented by Dr. C. P. Franklin.
10. Chinese head (model). Presented by Dr. Charles D. Hart.

The Mütter Lecture for 1912 was delivered on December 6 by Dr. Joseph Colt Bloodgood, of Johns Hopkins University, Baltimore, Maryland. Subject, "The Relation of Pathology to Surgical Diagnosis, with Special Reference to Border-line Pathological Conditions." Illustrated by a Lantern Slide Demonstration on Border-line Lesions of the Female Breast. There were a good many of the Fellows of the College present and a large attendance which quite filled the Cadwalader Room.

Dr. Clarence Hoffman, the Curator of the Museum, has continued to give his personal attention to the cataloguing of the contents of the Museum and in addition has placed individual cards in front of every separate specimen, with a statement of each.

This will be a useful attraction to those visiting the Museum—as the cards have also numbers of reference for fuller information which can be found in the catalogue.

Respectfully submitted,

George McClellan,
Chairman.

LIST OF PAPERS: SECTION ON OPHTHALMOLOGY

December, 1911

Concerning the Site of the Lesion in the Production of Ring Scotoma, by Dr. H. M. Langdon.

Report of a Case of Migraine with Ring Scotoma, by Dr. Wm. Zentmayer.

A Case of Keratitis Rosacea, by Dr. Burton Chance.

A Fatal Case of Streptococcic Bacteremia, with Panophthalmitis, by Dr. H. F. Hansell.

Exhibition of a Case of Metastatic Gonorrheal Iritis, Treated with Neisser Bacterin, by Dr. E. A. Shumway.

A Report of a Case of Antral Disease in a Four Months' Old Child, with Marked Orbital Symptoms, by Dr. Frederick Krauss.

Exhibition of a Patient with Polycoria Resulting from Atrophy of the Iris, by Dr. Charles R. Heed.

January, 1912

Double Optic Atrophy in a Case of Infantile Tabes, by Dr. George E. Price and Dr. Charles E. G. Shannon.

Intradural Tumor of the Optic Nerve, with Histologic Study, by Dr. George E. de Schweinitz.

Optic Atrophy Following Hemorrhage from the Stomach, by Dr. Wm. Zentmayer.

Exhibition of a Patient with Optic Atrophy and Scar of a Lacerated Wound at the External Canthus, the Result of Obstetric Injury, by Dr. Wm. Campbell Posey.

February, 1912

Cryptophthalmos and Congenital Ankyloblepharon; Report of Five Cases in one Family, with Exhibition of Patients, by Dr. Harold Goldberg.

Hereditary Deficiency of the Light Sense in an Otherwise Healthy Eye, with Exhibition of Henry's Photometer, by Dr. H. Maxwell Langdon.

A Case of Aspergillus Flavus Ulcer of the Cornea, Exhibition of Specimens, by Dr. Wm. Zentmayer.

Vossius Ring-shaped Opacity in the Lens, Following Contusion, by Dr. T. B. Holloway.

March, 1912

A Case of Interstitial Keratitis Following Traumatism, by Dr. William M. Sweet.

Secondary Glaucoma in Interstitial Keratitis, by Dr. Edward A. Shumway.

A Case of Melanotic Sarcoma of the Chorioid with Pathological Report, by Dr. Charles R. Heed. Exhibition of Specimens, by Dr. Sidney Olsho (by invitation).

Exhibition of a Transilluminator, by Dr. Charles R. Heed.

Exhibition of a Patient Showing the Results of a Blepharoplasty, Performed for the Relief of Anchyloblepharon and Complete Symblepharon of the Inner Third of the Lids, Following a Burn, by Dr. Wm. Campbell Posey.

April, 1912

A Case of Sarcoma of the Chorioid, with Exhibition of the Specimen, by Dr. John Turner.

Exhibition of a Case of Ectropion relieved by Snellen Sutures, by Dr. Wm. M. Sweet.

Optic Neuritis with Temporary Blindness due to Ethmoiditis, by Dr. Wm. Zentmayer.

A Case of Edema of the Orbits, Secondary to Facial Dermatitis, by Dr. Howard F. Hansell.

Demonstration of a Case of Secondary Glaucoma in Interstitial Keratitis, by Dr. E. A. Shumway.

Demonstration of the Result of a Fricke Flap Operation for Keloid Epithelioma of the Lid, by Dr. S. Lewis Ziegler.

Exhibition of the Zeiss Teleater Prism Glass with Loupe Attachment, by Dr. S. Lewis Ziegler.

Exhibition of a Boy with Probable Pseudoglioma, by Dr. Frederick Krauss.

Report of the History of a Case of Polycythemia with Choked Disks; Exhibition of the Patient, by Dr. Wm. T. Shoemaker.

Exhibition of a Scotometer, by Dr. H. M. Langdon.

October, 1912

A New Prism Test for Muscular Imbalance, by Dr. J. Thorington.

Severe Burn of the Eyeball from Explosion of a Zodiac Golf-ball; Exhibition of the Patient, by Dr. John T. Carpenter.

Exhibition of a Caustic Alkali Burn of the Eyes, Following the Explosion of a Golf-ball, by Dr. H. Maxwell Langdon.

An Unusual Form of Congenital Family Cataract, by Dr. T. B. Holloway.

Exhibition of a Patient Showing the Results of Ectropion Treated by Snellen Sutures, by Dr. Wm. M. Sweet.

November, 1912

Some Interesting Postoperative Complications and their Treatment, by Dr. George E. de Schweinitz.

A Case of Syphilitic Retinitis with Unusual Features, by Dr. Howard F. Hansell.

Some Experiences with Ganglion Anesthesia in Enucleations of the Eyeball, by Dr. Wm. M. Sweet.

Exhibition of a Patient Showing the Result of a Maxwell Operation for Restoration of the Cul-de-sac, by Dr. Wm. Zentmayer.

Exhibition of Nernst's Lamp and Bardsley's Sclerecotome, by Dr. S. Lewis Ziegler.

THOMAS B. HOLLOWAY,
Clerk.

LIST OF PAPERS: SECTION ON OTOLOGY AND LARYNGOLOGY

December 20, 1911

Presentation of Teratoma Removed from the Pharynx, by Dr. C. C. Eves (by invitation).

Report of a Case of Chronic Suppurative Otitis Media, with Unusual Complications; Operation, by Dr. Francis R. Packard.

Specific Lesions in the Nasopharynx and Tonsils, by Dr. James A. Babbit. Discussion by Drs. Uhle and MacKinney (by invitation).

January 17, 1912

Report of a Case of Carcinoma of the Antrum of Highmore, by Dr. George B. Wood. Presentation of patient.

Report of a Case of Postauricular Herpes, by Dr. Benjamin D. Parish. Presentation of patient.

Report of a Case of Herpes Zoster of the Ear, by Dr. Arthur A. Bliss.

Notes on Cases of Nasal Asthma, by Dr. Arthur Watson.

February 21, 1912

The Investigation of the Ethmoid and Sphenoid Sinuses in Refractory Cases of Postnasal Catarrh, by Dr. Charles P. Grayson.

Exhibition of Tonsils Weighing More than Thirty-two Grams, by Dr. Ben C. Gile (by invitation).

A Fatal Case of Acute Otitic Brain Abscess and Cerebrospinal Meningitis of Extreme Rapidity, by Dr. B. Alexander Randall.

Some Considerations in Reference to the Nasal Septum, by Dr. William R. Butt (by invitation).

March 20, 1912

The Anatomy and Relations of the Tonsils in Hardened Body, with Special Reference to the Correct Interpretation of the Plica Triangularis. The Principles and Practice of Tonsil Enucleation as Based Thereon, by Dr. George Fetterolf.

April 17, 1912

Presentation of Cases: (1) Patient with Hysteria Simulating Mastoid Disease; (2) Patient Illustrating Nature's Effort at Mastoid Exenteration, by Dr. S. MacCuen Smith.

Presentation of Two Cases of Killian Operation on the Frontal Sinus, with Complete Exenteration of the Ethmoid Labyrinth and Opening the Sphenoid Sinus, by Dr. E. B. Gleason.

October 16, 1912

Report of a Case with Abductor Paralysis of the Right Vocal Cord Due to an Osteosarcoma of the Mediastinum, by Dr. Ralph Butler. Presentation of patient.

Bismuth Paste in Ear and Nose Work, by Dr. George Coates.

November 20, 1912

Presentation of a Skull Showing a Dehiscence over the Lateral Sinus and Absorption of other Parts of the Base; Question as to Any Prior Operation, by Dr. B. Alexander Randall.

Report of a Tonsil Operation with Alarming Symptoms from Severe Acidosis, by Dr. George B. Wood.

Exhibition of a Series of Frozen Sections of the Head, by Dr. William R. Butt (by invitation).

Report of a Case of Brain Abscess, by Dr. B. Alexander Randall.

RALPH BUTLER,
Clerk.

LIST OF PAPERS: SECTION ON GENERAL MEDICINE

December, 1911

The Partially Developed Form of Hyperthyroidism; its Symptoms and Treatment, by Dr. E. J. G. Beardsley.

Pellagra in its Relation to Neurology and Psychiatry, by Dr. Beverly R. Tucker, of Richmond, Va. (by invitation).

Osteitis Deformans, with Report of Two Cases, by Dr. B. Franklin Stahl.

The Influence of Theophyllin on Nitrogen Excretion, by Dr. William H. Welker (by invitation) and Dr. C. B. Farr.

January, 1912

The Mackenzie Clinic, by Dr. James E. Talley.

The Functional Diagnosis of Pancreatic Disease, by Dr. Joseph H. Pratt, of Boston (by invitation).

February, 1912

Some New Facts Concerning William Harvey, by Dr. S. Weir Mitchell.

Exhibition of a Case of Acute Lymphatic Leukemia Showing a Large Mediastinal Mass, by Dr. J. Norman Henry.

Laceration of the Mesentery as a Cause of Intestinal Obstruction, by Dr. Joseph Sailer.

Report of a Case of Septicemia in a Child, Following a Mastoid Operation, by Dr. E. J. G. Beardsley.

March, 1912

A Case of Diabetes Mellitus and Tuberculosis of the Adrenal Glands, by Dr. Charles M. Montgomery.

Sarcoma of the Mediastinum, by Dr. Herman B. Allyn.

Sarcoma of the Lung, by Dr. A. A. Stevens.

Intrathoracic Tumors, by Dr. R. G. Torrey and Dr. Joseph Sailer.

Note on a Peculiar State of Asthenia of Short Duration, by Dr. David Riesman.

Improvement in the Method of Giving Artificial Nauheim Baths, by Dr. Myer Solis-Cohen.

April, 1912

Arguments against Tuberculin Therapy, by Dr. Joseph Walsh.

Personal Experiences with Tuberculin Treatment, by Dr. Lawrason Brown, of Saranac Lake (by invitation).

May, 1912

Some Notes on Methods of Determining the Pancreatic Function, by Dr. Alfred Stengel.

A Case Showing Phases of Heart Block and Auricular Fibrillation, by Dr. James E. Talley.

Personal Experiences with the so-called Vaccine Treatment of Acute Articular Rheumatism, by Dr. Edward H. Goodman.

October, 1912

A Case of Extrasystolic Arrhythmia Due to Insufficient Vagus Control, by Dr. George W. Norris.

Report of Several Cases of Continued Fever of Anomalous Character, by Dr. David Riesman.

Syphilis of the Lung, by Dr. H. R. M. Landis and Dr. Paul A. Lewis (by invitation).

Some observations on the Coagulation of Oxalated Plasmas, by Dr. O. H. Perry Pepper and Dr. J. Harold Austin (both by invitation).

November, 1912

Final Report on a Case of Contusion of the Chest Followed by Persistent Hemoptysis and Presystolic Murmur and Thrill, by Dr. J. C. Wilson.

Exophthalmic Goitre from the Standpoint of the General Practitioner, by Dr. Josephus T. Ullom.

Two Cases of Pneumonia with Recovery Occurring in Tuberculous Subjects, by Dr. R. Max Goepp.

Incidence of Purpura in Pulmonary Tuberculosis, by Dr. John M. Cruice.

LIST OF PAPERS: SECTION ON MEDICAL HISTORY

December 20, 1912

Dr. H. R. M. Landis. "Frederick Goll, the Discoverer of the Sensory Tract in the Spinal Cord."

Dr. Frederick P. Henry. "Asellius and the Discovery of the Lymphatic Circulation."

Dr. Richard M. Pearce. "Lantern Demonstration of the Illustrations of Early Treatises on Physiology."

H. R. M. Landis,
Clerk.

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